MASS MORTALITY OF BOTTLENOSE DOLPHINS IN 1987–88

HEARINGS

BEFORE THE

SUBCOMMITTEE ON
OVERSIGHT AND INVESTIGATIONS

OF THE

COMMITTEE ON
MERCHANT MARINE AND FISHERIES
HOUSE OF REPRESENTATIVES

ONE HUNDRED FIRST CONGRESS

FIRST SESSION

ON

THE CONCLUSIONS OF THE CLINICAL INVESTIGATION OF THE 1987-88 MASS MORTALITY OF THE BOTTLE-NOSE DOLPHINS ALONG THE UNITED STATES CENTRAL AND SOUTH ATLANTIC COASTS

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MASS MORTALITY OF BOTTLENOSE DOLPHINS IN 1987-88

TUESDAY, MAY 9, 1989

HOUSE OF REPRESENTATIVES,
SUBCOMMITTEE ON OVERSIGHT AND INVESTIGATIONS,
COMMITTEE ON MERCHANT MARINE AND FISHERIES,
Washington, D.C.

The Subcommittee met, pursuant to notice, at 2:17 p.m., in Room 1334, Longworth House Office Building, the Honorable Thomas M. Foglietta (Chairman of the Subcommittee) presiding.

Members present: Representatives Foglietta, Pickett, Pallone, Carper, Hughes, Manton, Schneider, Saxton, and Saiki.

Staff present: Marci Bortman, Phil Rotondi, Peter Marx, Lori Williams, Jim McCallum, Kurt Oxley, Jim Matthews, Christophe Toulou, Brook Ball, Chris Dollase, Nancy Tyson, and Mike Haas.

Mr. FOGLIETTA. The Subcommittee on Oversight and Investigations of the Committee on Merchant Marine and Fisheries will now come to order.

Allow me to first apologize for our tardy beginning, but as I am sure you know, we did have two votes in rapid succession which kept the Members over on the Floor.

So, with that, we will proceed with this hearing.

I am happy that we are all here today to discuss this problem.

OPENING STATEMENT OF HON. THOMAS M. FOGLIETTA, A U.S. REPRESENTATIVE FROM PENNSYLVANIA, AND CHAIRMAN, SUBCOMMITTEE ON OVERSIGHT AND INVESTIGATIONS

Mr. FOGLIETTA. The Subcommittee on Oversight and Investigations is here to receive testimony on the conclusions of the clinical investigation of the 1987-88 mass mortality of the bottlenose dolphins along the United States Central and South Atlantic Coasts.

On June 15th, 1987, the Marine Mammals Stranding Center in Brigatine, New Jersey reported that a bottlenose dolphin had washed ashore. This was not an unusual occurrence for the New Jersey coast, which averages three dolphin wash-ups per year. In fact, the center characterized this particular event by reporting simply that—and I quote: "The first bottlenose dolphin has washed ashore on the New Jersey coast for the 1987 season." End quote.

But all was not as it seemed. Over a 30-day period, from June to July 1987, 47 dolphins would strand in New Jersey. A record number of strandings would occur down the coast to Florida. Eleven months later, when this unparalleled event concluded, 744 bottlenose dolphin deaths had been recorded.

Normally, in any given season, we would have expected to record only 42 dolphin deaths. And experts on marine mammals estimate that 50 percent or more of the near-shore stock of the bottlenose dolphins was depleted. With a population reduction of this magnitude, it may take more than a century for the stock to return to pre-epidemic levels.

Public concern about the die-off was immediate. Our near-shore oceans carry our commerce and provide us with food and recreation. On a typical, sunny weekend day, approximately 10 million people are in, around, or on the East Coast beaches. Perhaps the mood of the public is best summed up by one headline which appeared near the height of the die-off in August 1987.

It read: "Before You Swim In The Ocean This Weekend, Read About The Mysterious Dolphin Deaths."

To investigate this extraordinary occurrence, the National Marine Fisheries Service, the Office of Naval Research, and the Marine Mammal Commission contracted with Dr. Joseph Geraci, a specialist—I hope I am pronouncing that properly, Doctor. Is it okay? It is an Italian pronunciation, I hope you would agree with that. —a specialist in marine mammal veterinary medicine, and professor of marine pathology at Ontario Veterinary College, University of Guelph, to head the interdisciplinary team of scientists from the National Oceanic and Atmospheric Administration, the United States Department of Agriculture, the Smithsonian Institution, the Environmental Protection Agency, and dozens of universities and private agencies.

What baffled and was of particular concern to scientists, as well as to those of us outside the scientific community concerned about our oceans and environment, was that there appeared to be no obvious cause or immediate trend to explain the mass mortality. The epidemic was non-selective, killing dolphins of both sexes and of all ages.

Dr. Geraci's 18-month clinical investigation is now concluded. The investigation finds, in layman's terms, that the dolphins died because of a naturally occurring toxin from "red-tide" algae.

Disasters like this happen swiftly and with no forewarning. Scientists do not have the necessary time to properly assess the situation in many cases. That is why it is vital that we learn as much as possible about the probable causes and solutions, so that we can prevent it from ever happening again.

If a man-made chemical, or the water quality contributed in any way to the death of the dolphins, we must address that larger problem. The fate of the entire ocean community is dependent on clean and safe water. It must be determined if the whole oceanic community is at risk.

It is the job of the Subcommittee on Oversight and Investigations to make sure everyone is putting forth their best effort to ensure the survival of future generations of dolphins and all marine life.

With today's hearing, the Subcommittee wants to review the report's conclusions, the methodology by which the conclusion was reached, and to explore possible alternative scenarios.

This hearing, I want to assure you, is not an adversarial proceeding. This fact, however, will not mitigate the intensity with which this Subcommittee intends to pursue this issue.

This mass mortality is, to use Dr. Geraci's own description, "the most extraordinary saga of cetacean disease on record."

There is a misconception that the report we are reviewing today answers all our questions about the die-out mystery. It does not, and I do not believe Dr. Geraci and I disagree on this point. Tough questions remain to be addressed, particularly the contributing role of pollution.

Did man-made contaminants in our ocean affect the natural resilience of these dolphins and render them more susceptible to the toxin and microorganisms that eventually killed them?

Our purpose today, then, is not to close the door on this tragic episode, but to put this extensive effort into context so that we can open the proper door for continued investigation and eventual remedy, if one is called for. Our marine life and our oceans are simply too important to all of us for us to do otherwise.

Mr. FOGLIETTA. I would like to recognize the distinguished Ranking Minority Member, a true leader in environmental issues, Congresswoman Schneider.

OPENING STATEMENT OF HON. CLAUDINE SCHNEIDER, A U.S. REPRESENTATIVE FROM RHODE ISLAND

Ms. SCHNEIDER. Thank you very much, Mr. Chairman. I would like to begin by thanking my colleague, Mr. Saxton, for initially bringing this report to my attention, and also, I would like to thank you very kindly for your speedy response in calling this hearing together.

From late June through May 1988, 742 Atlantic bottlenose dolphins washed ashore on the beaches from Mr. Saxton's district all the way down to Florida, and probably thousands of others died offshore. And perhaps as much as half of the inshore bottlenose dolphin population perished during this particular epidemic.

I think, however, that we need to put all of this in perspective, and to recognize that in any average year, less than 50 dolphins wash up on Atlantic beaches. During this particular time period, there were innumerable other events in the Atlantic which indicated that things were not as they should be.

As we look at what was going on at that time, we see that in the fall of 1987, 13 humpback whales washed ashore on Cape Cod. During 1987 and 1988 beaches were closed from my own State of Rhode Island all the way to North Carolina due to medical waste, garbage, and human waste washing ashore. There were also very large fish kills which occurred in 1987 and 1988 in Long Island Sound, and also off the New Jersey coast, not to mention the fact that "brown tides" have eliminated a once lucrative commercial and recreational shellfish industry in Peconic Bay off of eastern Long Island.

And the 106-mile dump site was officially opened to sewage sludge dumping on May 17th, 1986. Needless to say, when you add up all of this—and even on the other side of the Atlantic Ocean there are some similar problems.

Last year, more than 15,000 harbor seals died in the North Sea. High levels of contaminants in their bodies weakened their immune systems and caused them to be susceptible to a viral infection.

As one of my constituents, and a well-recognized international scientist, Dr. Smayda, will later point out in his testimony, it may not be a coincidence, the drastic increase in the number, size, and severity of phytoplankton blooms in the past 15 years coincides with the increase of global problems such as acid rain or the "greenhouse effect," increased ozone-layer destruction; deforestation, and also coastal eutrophication.

Many of the dolphins that washed ashore during this epidemic had increasingly high levels of contaminants. One specimen had 6,800 parts per million of PCB in its liver. Now it is important to keep that all in context because the FDA's acceptable level for human consumption is 2 parts per million.

So, with the conglomeration of pesticides, chemicals, garbage, and human wastes, that we continually pour, pipe, pump, and barge into the Atlantic Ocean every day, I find it hard to believe that this could not have been, at the very least, a contributing factor to the bottlenose dolphin epidemic.

And I thank you, Mr. Chairman, for giving us the opportunity to investigate this more thoroughly.

Mr. FOGLIETTA. Thank you. Congressman Pickett, 2nd District of Virginia.

Mr. PICKETT. Mr. Chairman, I will submit a statement for the record, with unanimous consent, please.

[The statement of Mr. Pickett follows:]

PREPARED STATEMENT OF HON. OWEN B. PICKETT, A U.S. REPRESENTATIVE FROM VIRGINIA

Mr. Chairman, I commend you for holding this hearing on what is undoubtedly one of the most perplexing and tragic environmental disasters in recent memory. Anyone who has spent time on our Mid-Atlantic beaches, and watched schools of bottlenose dolphins rolling gracefully in the surf, certainly has to appreciate the scope of this tragedy. The testimony we will receive today will show that some 2,500 dolphins, or 50 percent of the near-coastal, migratory stock may have perished during the 1987 epidemic. It will be many years before this population of dolphins is fully replenished.

Today, we have with us Dr. William Evans, the Undersecretary of Commerce for Oceans and Atmosphere, and Dr. J.R. Geraci, the Government's principal investigator into this epidemic. Dr. Geraci's finding were reported earlier this year and attributed the epidemic to brevetoxin, a naturally occurring toxin found in connection with red tide algae. Dr. Geraci's investigation was headquartered in Virginia Beach, and based on what I read about that investigation in the local press, I don't think there is any doubt that the Geraci team worked diligently and in good faith to get to the bottom of this.

The questions I have this morning center on the scope of the investigation and some of the methodology used. None of us has any interest in attacking the efforts that have been made thus far; rather, we simply want to make certain that the Government not rush to conclusions without considering all of the relevant factors.

Some of those factors include: (1) We need a more detailed explanation of the extent to which man-made pollutants may have contributed to this epidemic. As this Subcommittee well knows, the summer of 1987 was a year in which several of our Mid-Atlantic beaches were fouled with medical wastes. It was also the year in which the 106-mile sewage dump site was opened, and we need to know whether these and other pollutants lower the dolphins' susceptibility to this toxin; (2) Several legitimate questions have been raised about the report's methodology, specifically whether adequate numbers of dolphins were tested; (3) The peer review process utilized during this investigation; and (4) Why other red tides have not had this effect upon marine mammals.

Again, I commend you Mr. Chairman for holding this hearing and for raising these questions. In my judgment, this episode is simply too important from an environmental standpoint for anything less than a searching review of the agency's procedures.

Mr. FOGLIETTA. Congressman Saxton. I would like to note for the record that Congressman Saxton has been very, very active on this issue since this epidemic began in June of 1987.

In fact we hear that your association with this issue began as a personal one. Is that correct?

Mr. SAXTON. Well, it did, as a matter of fact. I was not swimming with dolphins, I was boating with them, and I saw one of the first that was on its way to wash up on the shore. That is correct.

OPENING STATEMENT OF HON. JIM SAXTON, A U.S. REPRESENTATIVE FROM NEW JERSEY

Mr. SAXTON. Mr. Chairman, let me express my appreciation to you, and the ranking Member, Miss Schneider, for your thoughtfulness in calling this hearing and giving consideration to a matter which I believe deserves a great deal of attention, and I believe that this subject may have far-reaching implications as to the health and welfare of our oceans.

Whether the epidemic of dolphin deaths along the East Coast in 1987 and 1988 remains a mystery or not in the aftermath of this hearing—and I suspect that it may, to some degree—the fact remains that we are confronted with a very worrisome phenomenon.

This hearing will hopefully provide us with an opportunity to investigate the need for additional congressional assistance or congressional action.

It may indicate to us the needs of the community of marine sciences as well, something that we may pay a great deal more attention to. And I do hope that the community of marine sciences will continue to collectively address this matter, because as far as I can see, the answers have yet to be found.

Dr. Geraci has estimated that as much as 50 percent of the Atlantic Coast's migratory stock of dolphins has been lost. That, to me, is tragic, and I believe there are few people who will challenge that assessment.

For the record, I am not a scientist, nor are most of the Members of my staff, even though they are very talented. However, we have read extensively the work of NOAA's task force, its conclusions, as well as the comments submitted by various marine scientists.

And although my heart would like to believe today that this event was a result of naturally-occurring phenomenon, I remain to be persuaded. I have brought with me a chart today, which I would like to use, which I believe poses some very serious questions.

The first bar, in blue, represents the 740 dolphins that reportedly washed ashore and were stranded during the 11-month period that this event took place. This is a conservative estimate, but it is what were found and what we can count. We do not know how many actually died, but it could have ranged into the thousands.

The second bar, in yellow, indicates the number of dolphins that were sampled in one way or another according to the report, and 347 dolphins appear in that column, and they were sampled by the NOAA study, for the NOAA study, in one way or another.

The third bar, shown in green, represents 83 dolphins tested for chlorinated hydrocarbons such as PCBs, DDT, and DDE. All dolphins contained in this category, incidentally, were found to contain contaminants that they were tested for—100 percent.

Fourth, and finally, the fourth bar in red and white shows the number of dolphins that were tested for brevetoxin. The red portion represents eight dolphins that tested positive for this toxin. These eight represent 1.1 percent of the total 740 that were found.

So that the task force appears to have drawn a conclusion, that based on these eight dolphins that tested positive for brevetoxin, out of the total 17 tested for that substance, that this phenomenon, these 740 deaths that we can count—and perhaps thousands more—were blamed on brevetoxin as a result of the red tides that were apparently off North Carolina, Virginia, and on south.

The 83 that were tested for other types of toxins like DDT and PCBs of various kinds, and DDE, seem to escape the conclusion that this could have been the factor which caused the deaths. This, to me, raises a very, very serious question.

I believe the point must be made that the number of dolphins that were reported with lesions and various degrees of degradation to the skin—I am told that there is documented evidence that the correlation between lesions and the effects of hydrochlorides do exist. I am likewise told that there is a lack of evidence of any correlation between brevetoxin poisoning and the appearance of lesions.

Be that as it may, as the chart very clearly points out, only eight of 17 dolphins contained brevetoxins. And so as a layman, I have to ask if it is fair and proper to conclude that brevetoxin was the culprit in the tabulated deaths of the 740 dolphins.

I know that the NOAA report concludes that the lesions resulted from other causes to which the dolphins became vulnerable. I know that it also attributes to this sublethal exposure to brevetoxin, but, again, I must ask if any studies can confirm this finding.

As probably everyone knows, the first dolphin to wash ashore was in New Jersey. The Chairman pointed that out a few minutes ago.

In New Jersey, however, there was no red tide in June or July. In fact the red tide reported further to the south did not migrate north until the fall of 1987, after the peak of the die-offs. Perhaps the dolphins could have migrated through that red tide—I do not know—but it serves to raise further questions about the migratory patterns of dolphins, and what their actual exposure to the "red tide" and the brevetoxin may have been. I hope our witnesses today can address this issue as well.

Finally, I do think it essential that our work continue, and that our initial study sponsored by the gentleman from Delaware, Mr. Carper, and others, be continued. It should be continued, not just for the post-mortem analysis, but to insure that there is expanded investigation into other possible causes of the epidemic.

The question of water quality, in particular, should be closely further examined, and, with that, Mr. Chairman, I look forward to hearing from the distinguished witnesses, and again, thank you for the opportunity to be here and take part in this hearing today.

[The statement of Mr. Saxton follows:]

Prepared Statement of Hon. H. James Saxton, a U.S. Representative from New Jersey

First, I want to express my appreciation to you, Mr. Chairman, for holding this hearing, and giving consideration and attention to a matter which, I believe, may hold far-reaching implications as to the health of our oceans.

Whether the epidemic of dolphin deaths along the East Coast remains a mystery or not in the aftermath of this hearing, the fact remains that we are confronted with a horrifying phenomenon.

My first personal exposure to this tragedy was totally unrelated to legislative business. I was sailing off the coast of Atlantic City with my son when we were both shocked and saddened to see a dead dolphin rolling in the waves.

This hearing will provide us an opportunity to investigate the need for any congressional assistance that may be required by the community of marine sciences, and I hope that the community of marine sciences will collectively address this matter.

Dr. Geraci, I thank you for coming today. I won't mince words—I am sure you are aware that your conclusions have created a stir of disbelief and criticism.

I admire your courage for standing up to that criticism and want to personally let you know my feelings are only those of confusion and the need for cooperation and understanding.

I am not a scientist, nor are members of my staff. However, we have read extensively the work of your task force, your conclusions, as well as the comments submitted by various marine scientists.

Although my heart would like to believe that this event was the result of a naturally occurring toxin, I must be candid and say that I remain to be persuaded.

A number of dolphins were reported with lesions, and various degrees of degradation to the skin too atrocious to mention. I am told there is documented evidence on the correlation between lesions and the effects of organochlorine compounds. I am likewise told there is a lack of evidence for any correlation between brevetoxin poisoning and the appearance of lesions.

Be that as it may, only 8 out of 17 dolphins contained brevetoxin—in varying amounts. And so, as a layman, I have to ask if it is fair and proper to conclude that brevetoxin was the culprit in the tabulated deaths of 740 dolphins.

I know in your report that you conclude that the lesions resulted from other causes to which the dolphins became vulnerable. I know that you attribute this to a chronic, sublethal exposure to brevetoxin. But again, I must ask if any studies can confirm this finding.

As probably everyone knows, the first dolphin to wash ashore was located in New Jersey. In June and July of 1987, however, New Jersey was not experiencing a red tide. According to reports I have received, the occurrence of fish deaths or shellfish poisoning normally associated with a severe red tide is not evident.

There was, in fact, a red tide reported in South Carolina. But it did not migrate up the coast until December of 1987, after the peak of the dolphin die-offs occurred in August.

Given these few items, I think one can readily understand why a layman, a non-scientist, would ask questions.

I think it is also essential that our work continue, and that the initial study sponsored by the Gentleman from Delaware, Congressman Carper, be continued. It should be continued, however, in the manner it was originally intended to pursue—a multi-agency approach, with the participation and sharing of information available from the various non-governmental agencies and organizations pursuing similar efforts in the marine sciences. And that the question of water quality—in particularly—be re-examined.

With that, I thank the Chairman, and I look forward to hearing from these distinguished scientists who have joined us here today.

Mr. Foglietta. I thank the gentleman. I recognize now the distinguished gentleman from New Jersey's 3rd congressional district, Mr. Frank Pallone. Congressman Pallone has been the leader in this issue since his election last November, and was among the first to express an interest in having this particular hearing. Congressman.

OPENING STATEMENT OF HON. FRANK PALLONE, A U.S. REPRESENTATIVE FROM NEW JERSEY

Mr. PALLONE. Thank you, Mr. Chairman. First, I would like to commend you and the Members of the Subcommittee, in particular, my colleague from New Jersey, Congressman Saxton, for convening this hearing to focus greater attention on one of the most devastating environmental events to strike the Atlantic Coast in recent years.

On February 1st of this year, NOAA held a press conference and issued a news release claiming to have arrived at a definitive conclusion on the cause of the mass die-off of dolphins. The dolphins, we were told, were poisoned by eating fish tainted by a naturally-occurring toxin from "red tide" algae.

Any possible role played by non-naturally-occurring pollutants were apparently dismissed. Unfortunately, the supporting evidence to back up this surprising hypothesis was not made available. At the time of the press conference on February 1st, we were told to expect a final report within a matter of weeks.

I received my copy of the report on April 26th, nearly three months after the press conference. The time lag between the first public announcement about the investigation's findings, and the issuance of the final report was extremely damaging to the credibility of NOAA, in my opinion, and cast suspicions about the validity of the findings of this investigative team.

However, after reading the report within the last couple weeks, I became even more dismayed. Some time between the February 1st press conference and the drafting of the final report, the tone grew far less definitive. The report acknowledges that the stricken dolphins lived in a very polluted environment yet the role of these pollutants is not given extensive consideration.

What the reader comes away with from this final report is that in blaming the deaths on brevetoxin, the investigators have a hypothesis, maybe even a plausible hypothesis, but they do not have a conclusion.

The study was hampered by severe limitations in terms of the scope and breadth of the investigation, and my criticism does not necessarily reflect so much on Dr. Geraci—I want that to be clear—but on the agency, NOAA, for failing to bring a wider range of disciplines and specialties into the investigation.

Why, for example, was the proven link between man-made substances such as PCBs and the symptoms observed on the dolphins overlooked, or disregarded? And why did the investigators make use of such a tiny sampling of dolphins found stranded on New Jersey beaches, given the fact that New Jersey is where the mortality was first observed, and where a significant proportion of the dolphins were stranded?

The last sentence of the report that we have before us today leads me to the conclusion that PCBs or chemical contaminants were just as likely to have been the cause that broke down the dolphins' immune systems and made them susceptible to red tide.

In my opinion, the report cries out for a reopening of the investigation, or a follow-up of the investigation to broaden the scope so as to look at the extent to which pollution may have contributed to the epidemic, either by breaking down the dolphins' immunity, or even by causing the massive red tide which supposedly was the cause of their death.

I also think that it is not a coincidence that the dolphin deaths occurred during the worst 2 year or 18-month period in the history of Atlantic Coast pollution problems, as has been alluded to by some of the other speakers here today.

I also feel that dolphins, historically, and certainly even in mythology, help people and relate to people. I think that what is happening here is that dolphins are really symptoms of what may be a larger pollution problem that ultimately may even affect humans. I think that in effect they are crying out for us to listen, and to see what the real problem is. We have to get to the bottom of it, and hopefully we will today. Thank you, Mr. Chairman.

[The statement of Mr. Pallone follows:]

PREPARED STATEMENT OF HON. FRANK PALLONE, JR., A U.S. REPRESENTATIVE FROM NEW JERSEY

Mr. Chairman, I would like to commend this Subcommittee, and in particular my colleague from New Jersey, Congressman Saxton, for convening this hearing to focus greater attention on one of the most devastating environmental events to strike the Atlantic Coast in recent years. In June 1987, unprecedented numbers of Atlantic bottlenose dolphins began washing ashore on the New Jersey coast. Within several months the dolphin strandings were taking place on hundreds of miles of beaches up and down the East Coast, and estimates are that at least half of the East Coast migratory dolphin population was lost. Responding to this crisis, Congress mandated the National Oceanic and Atmospheric Administration (NOAA) investigate the cause. We gather this afternoon in the hope of arriving at a better understanding of how this year-and-a-half-long investigation was conducted, why its final hypothesis was chosen, and why other potential causes were disregarded.

On February 1st of this year, NOAA held a press conference and issued a news release claiming to have arrived at a definitive conclusion on the cause of the mass die-off of dolphins. The dolphins, we were told, were poisoned by eating fish tainted by a naturally occurring toxin from red tide algae. Any possible role played by non-naturally occurring pollutants was apparently dismissed. Unfortunately, the supporting evidence to back up this surprising hypothesis was not made available. At the time of the press conference, we were told to expect the final report within a matter of weeks. I received my copy of the report on April 26, nearly three months after the press conference.

The time lag between the first public announcement about the investigation's finding and issuance of the final report was extremely damaging to the credibility of NOAA, and cast suspicions about the validity of the findings of this investigative team—however highly regarded Dr. Geraci and his associates may be in the scientific community. Indeed, the delay was probably one of the major factors leading to the calling of this Oversight and Investigations hearing.

However, after reading the report, I became even more dismayed. Some time between the February 1st press conference and the drafting of the final report, the tone grew far less definitive. The report acknowledges that the stricken dolphins lived in a very polluted environment, yet the role of these pollutants is not given extensive consideration. What the reader comes away with from this final report is that in blaming the deaths on brevetoxin, the investigators have a hypothesis, maybe even a plausible hypothesis, but they do not have a conclusion.

While I may lack the background to fault the science or methodology of the report, I can say with confidence that the study was hampered by severe limitations in terms of the scope and breadth of the investigation. This criticism does not necessarily reflect so much on Dr. Geraci as it does on NOAA for failing to bring a wider range of disciplines and specialties into the investigation.

Why, for example, is so little attention devoted to the very complex behavioral patterns of dolphins in the discussion of the role of communicable disease? Why were there no attempts to study the role of unusual currents and above-average water temperatures during the period in question? Why was there such a notable failure to tie the dolphin deaths in with changes in ocean dumping practices—notably the increased use of the 106-mile sewage sludge dump site—or any other isolated dumping, discharges or other events in the ocean? Why was the prove link between man-made substances such as PCBs and the symptoms observed on the dolphins overlooked or disregarded? And why did the investigators make use of such a tiny sampling of dolphins found stranded on New Jersey beaches, given the fact that New Jersey is where the mortality was first observed and where a significant proportion of the dolphins were stranded? Finally, perhaps the most important question is why were these factors not even acknowledged as severe limitations to a truly comprehensive investigation?

I also believe that Congress and the general public should be quite disturbed to note that the initial announcement that red-tide brevetoxin killed the dolphins was made with tremendous fanfare, while this final report with its notable limitations and uncertainties was so quietly (and tardily!) brought to the attention of the Members of this Committee.

In sum, I believe that the brevetoxin-red tide finding represents not so much a theory as a hypothesis—an educated guess. Much more work is required to arrive at a conclusive solution to this very disturbing mystery. We do the public less of a disservice by admitting we do not know the answer than to provide the public with an "answer" that leaves great room for doubt.

I look forward to the testimony of Dr. Evans, Dr. Geraci, and the other panelists, in addressing these concerns and those of the other Members of the Committee.

Mr. FOGLIETTA. Thank you. Congressman Hughes has been a leader of a host of environmental issues and is the architect of last year's legislation to end ocean dumping. I am happy to have him here today, and I would ask Congressman Hughes to make a statement, if he has one.

OPENING STATEMENT OF HON. WILLIAM J. HUGHES, A U.S. REPRESENTATIVE FROM NEW JERSEY

Mr. HUGHES. Well thank you very much, Mr. Chairman. I want to congratulate you and the Ranking Minority Member for convening this hearing, and my colleagues from New Jersey for focusing attention upon this tragedy that occurred in 1987-1988.

Mr. Chairman, I have a statement which I am going to insert in the record. We have a long hearing list. I am not going to read it. I just want to tell you, Mr. Chairman, that after reading the report, there are more questions raised than are answered, and I am dismayed, because I just cannot imagine why the report overlooked many of the man-made substances that we deposit in the ocean.

The timing of the dumping—which began, as I recall, in May of 1987—did not reach the 100 percent mark until January 1 of 1988, but it is during this period of time when we began to see the mortality rate increase significantly in the dolphin population.

So I am anxious to hear the witnesses, and some of the explanations that might be offered today, and hopefully, we, too, can get to the bottom of just what occurred. Thank you, Mr. Chairman.

[The statement of Mr. Hughes follows:]

PREPARED STATEMENT OF HON. WILLIAM J. HUGHES, A U.S. REPRESENTATIVE FROM NEW JERSEY

Mr. Chairman, thank you for inviting me to join you today in hearing from the witnesses on the clinical investigation of the 1987 to 1988 mass deaths of bottlenose dolphins along the Atlantic Coast.

I appreciate the efforts of Dr. Geraci, the National Marine Fisheries Service, the Navy, the Marine Mammal Commission, and others who have worked on this report in attempting to determine the cause of some 740 known dolphin deaths that have occurred.

The conclusion of the report—that the dolphins were poisoned by brevetoxin, or the Red Tide organism—indicates an unprecedented phenomenon that is natural, widespread, and has the potential to recur.

However, after reading the report, I have some serious questions with regard to whether sufficient consideration has been given to the possibility that pollution weakened the dolphins, making them susceptible to bacteria and eventually leading to the die-offs.

I also have questions about the cause of the Red Tides. Are our oceans more susceptible to frequent and widespread occurrences of Red Tide blooms as we continue to pollute our waters with sewage sludge, industrial discharges, combined sewer overflows, and nonpoint source pollution?

Soon after dead and dying dolphins began washing up along the coast, it was clear that we knew very little about these mammals—let alone the cause of the dolphin deaths that were occurring in such epidemic proportions.

Mr. Chairman, it is important that we support dolphin research and pursue our investigation of the dolphin deaths. We also need to support studies on Red Tide blooms and their relation to marine pollution. Just as importantly, we need to develop better baseline data so that we might better assess the condition of our oceans. Only then may we be able to answer many of the unknowns that still exist and, if possible, prevent a reoccurrence of the dolphin tragedy.

Thank you and I look forward to hearing from today's witnesses.

Mr. FOGLIETTA. I know Congresswoman Saiki is interested in this particular issue, and do you have any statement?

OPENING STATEMENT OF HON. PATRICIA SAIKI, A U.S. REPRESENTATIVE FROM HAWAII

Mrs. SAIKI. Thank you very much, Mr. Chairman. I do appreciate being invited to attend this hearing over an issue with which I share concern with all of you.

The dolphin deaths, of course, along the Atlantic Coast is rather devastating, and I know that in listening to the experts today maybe we can come to some conclusions. But I know that the concern we have for man-made pollutants in the ocean can be extended to, perhaps, man-made pollutants in man-made lagoons.

We had recently two dolphin deaths at the Waikoloa Hyatt on the Big Island of Hawaii. I feel personally responsible because I helped to vote through the amendment to allow for dolphins to be on display in resort pools, and also to, in a way, endorse—not quite fully—but give permission to allowing swimming with the dolphins. And I hope that the agency will provide me with some answers as to the causes for those two dolphin deaths, and maybe through their information, and our investigation through this Committee, we can prevent future deaths in man-made situations such as resorts and hotel pools. Thank you.

Mr. FOGLIETTA. I thank the gentlelady. Congressman Carper is the author of last year's amendment to the Marine Mammals Protection Act on mass mortality of the bottlenose dolphins and he is here with us today. Congressman Carper.

OPENING STATEMENT OF HON. THOMAS R. CARPER, A U.S. REPRESENTATIVE FROM DELAWARE

Mr. CARPER. Thank you, Mr. Chairman, and to Members of the Subcommittee, thank you very much for holding these hearings, and for permitting those of us who do not serve on this Subcommittee to be here today to join in the hearing.

I must say, candidly, that I have some serious misgivings about the report that is being discussed. I have some serious misgivings about its scope and about its methodology.

There appears to be a prejudice in this report to promote a single, and I think, highly-contentious theory for the die-off that has been discussed already, while discounting other equally viable possibilities.

As the author—you mentioned, Mr. Chairman—as the author of a provision which I think everyone sitting here who was a Member of the 100th Congress—as the author of that provision in last year's Marine Mammal Protection Act reauthorization to provide further direction to the National Marine Fisheries Service regarding this study, I am particularly interested in hearing from our witnesses today on their assessment of this report.

The Carper Amendment requires a full investigation of the die-offs, causes and effects, with specific attention paid to the role pollution may have played.

Now, Mr. Chairman, earlier this year, NMFS indicated to me, and to other members of this panel, that the study before us today should meet the requirements set forth by the Carper Amendment. I have concluded—and I suspect that many of my colleagues here today will conclude—that that is not the case.

I would suggest that glaring omissions and questions persist in our knowledge of what happened off of our shores. This report points to as much in its conclusions, and recommends that continued study is still in order.

Mr. Chairman, on the first of this month, I sent a letter to Mr. James Brennan, the Assistant Administrator for Fisheries at NOAA, and I asked that the questions posed by the Carper Amendment be adequately addressed, and with the indulgence of this Subcommittee, Mr. Chairman, I would like to submit this letter for the record and I would ask unanimous consent to do so.

Mr. FOGLIETTA. So ordered.

[The letter may be found at end of hearing.]

Mr. CARPER. In that letter, I have asked that NMFS report back to this Subcommittee, and to the Senate Commerce Committee by January 1 of next year on what continuing activities NMFS will undertake in coordination with other public and private agencies to further resolve these remaining critical issues.

And I would suggest that the Committee on Merchant Marine and Fisheries follow up on this request, and I will work with my colleagues here and on the Full Committee to do just that.

Let me say, in closing, to everyone at this hearing today, that we have a responsibility not to sensationalize the potential causes of the dolphin die-off.

I do not welcome the possibility of headlines indicating that PCBs in our coastal waters killed these dolphins. We do not know anything of the sort. But having said that, it is essential that we develop a clear understanding of what is going on in our coastal waters, if we are to take appropriate action to protect these waters, and ourselves.

Failure to do so would be to abdicate a major responsibility we have to protect our marine environment, the natural resources on which we depend so much, and the well-being of all who depend on a healthy and a vibrant ocean.

Again, Mr. Chairman, thank you for holding these hearings, and thank you for giving us all a chance to participate.

[The statement of Mr. Carper follows:]

PREPARED STATEMENT OF HON. THOMAS R. CARPER, A U.S. REPRESENTATIVE FROM DELAWARE

Mr. Chairman and Members of the Subcommittee, I appreciate the opportunity to join you today to review the National Marine Fisheries Service (NMFS) report on the dolphin die-off off the Atlantic Coast in 1987 and 1988.

As the author of a provision in last year's Marine Mammal Protection Act reauthorization to provide further direction to the National Marine Fisheries Service regarding this study, I am particularly interested in hearing from our witnesses their assessment of this report. My amendment required the Secretary of Commerce—in which NMFS is located—to examine: (1) the cause or causes of the die-off; (2) the effect of the die-off on inshore and offshore populations of bottlenose dolphins; (3) the role played by pollution in the die-off; (4) the extent to which other species of marine mammals were affected; and (5) any other matters regarding the causes and effects of the die-off.

The amendment specifically required that the study be done in consultation with other Federal agencies (including the Environmental Protection Agency), the Smithsonian Institution, State agencies, universities, and foreign agencies and institutions, including any that were involved in the investigation of the 1987–88 seal die-off in the North Sea. A report on the findings of this expanded study were to be forwarded to the relevant House and Senate Committees by January 1, 1990.

Despite a letter sent to this Committee earlier this year suggesting that these issues had been adequately addressed in the study before us today, I would suggest that glaring omissions and questions persist in our knowledge of what happened off our shore. The report points this out, and recommends that continued study of these questions is in order.

Mr. FOGLIETTA. I thank the gentleman. Now Florida has more experience with dolphins than any other area of the country, so the Subcommittee welcomes Congressman Porter Goss of Florida's 13th congressional district.

OPENING STATEMENT OF HON. PORTER GOSS, A U.S. REPRESENTATIVE FROM FLORIDA

Mr. GOSS. Thank you, Mr. Chairman. It is true that dolphins are part of our quality of life in Florida, and it is equally true that "red tide" is part of our way of life in Florida, and we have learned to live with both.

I would like to submit for the record a report from Dr. Richard Pearce, which involves findings on red tide and dolphins that he has recently completed. He has provided testimony to NOAA on it.

We have, for some years in Florida, in our coastal communities in the State, through marine, and other resources that are well accredited, and certainly capable of doing the job that they have been doing, been trying to unlock some of these mysteries, and I believe this report would be useful to this Committee and I would like to have it entered into the record.

I look forward to the testimony today and I appreciate you including me in this.

[The report submitted by Mr. Goss can be found at end of hearing.]

Mr. FOGLIETTA. Without objection the report will be included in the record and we thank the gentleman.

Mr. FOGLIETTA. Congressman Tallon of South Carolina.

OPENING STATEMENT OF HON. ROBIN TALLON, A U.S. REPRESENTATIVE FROM SOUTH CAROLINA

Mr. TALLON. Mr. Chairman, thank you. As we all know, I am not a Member of the Subcommittee, and I just appreciate so much you and Miss Schneider, and the Members of the Subcommittee for letting some of us that have a great interest in this sit in, and participate in the hearing here today.

Mr. Chairman, we read so many reports, and hear so many statistics bandied about in the Congress, and sometimes they tend to run together, but there is one set of numbers that I cannot forget—750 dead dolphins, 55 washed up on the beaches of South Carolina, and certainly the possibility and potentiality of thousands more dead at sea that were never discovered.

I, along with the rest of the Subcommittee, am anxious to hear from Dr. Geraci and members of the panel on the factors considered in the bottlenose dolphin investigation. In particular, I am interested in the research on pollution as a factor.

The report mentions pollution, but fails to demonstrate why the "red tide" should be considered the cause when significant levels of man-made pollutants were also found to be present in the dolphins tested.

It seems to me that this country—and I think we have heard it from other Members of the Subcommittee this afternoon—is learning the hard way, that we cannot disregard pollution.

From the oil-covered Prince William Sound to medical wastes found off the New Jersey shore, we are slowly but surely learning that our pollution problems are entirely too pervasive to ignore.

If you do not ask the right questions you are never going to get the right answers, Mr. Chairman, and we know that hastily, ill-founded conclusions on a matter like this can be deadly.

Let's ask enough questions. Let's ask the right questions. We are trying to get at the truth here. There are certainly valid concerns, and I think valid questions about the NOAA report.

Again, Mr. Chairman, thank you for holding these hearings.

Mr. FOGLIETTA. I thank the gentleman. Congressman Manton has been very involved in ocean issues, and last year was a key participant in the Ocean Dumping Ban Act legislation. Congressman Manton of the 9th congressional district of New York.

OPENING STATEMENT OF HON. THOMAS J. MANTON, A U.S. REPRESENTATIVE FROM NEW YORK

Mr. MANTON. Thank you, Mr. Chairman, for allowing me to participate in today's hearing even though I am not a Member of your Subcommittee. I also commend you for holding this important hearing on the tragic dolphin deaths which were experienced along the Atlantic Coast in 1987 and 1988.

The unusual number of dolphin deaths has apparently ceased. It is important, however, for the Congress to determine the exact cause of these deaths. We need to learn what, if anything, Congress can do to prevent such a mass mortality from ever occurring again. I am particularly concerned about assertions pertaining to the validity of the findings published in the final report on the dolphin mortality issued by the National Oceanic and Atmospheric Administration.

If the concerns over the methodology and the findings are correct, we must begin a new round of inquiries to ascertain the cause of these deaths. We must also ensure adequate steps are taken to protect these beautiful and intelligent cetaceans. Clearly, if these deaths cannot be attributed to natural causes, then whatever caused the mass mortality of these dolphins undoubtedly will continue to destroy the East Coast dolphin population, and, ultimately, the marine environment as a whole.

Mr. Chairman, I was encouraged by the preliminary announcement by NOAA that this epidemic was a result of a naturally-occurring phenomenon, an unusual episode of the so-called "red tide," and not the result of pollution. Recently, I have begun to hear arguments against this single-cause theory. Critics claim pollutants may indeed have played a major role in this deadly epidemic. However, I do not believe scientists critical of the report's conclusions have yet to adequately demonstrate that pollutants, rather than the naturally-occurring biotoxin, caused these deaths.

Mr. Chairman, regardless of the outcome of this debate over the report, one pressing point is quite clear in my mind. Our near-coastal waters are severely distressed. These waters continue to receive vast quantities of pollutants daily. We need to address these pollution problems expeditiously, particularly our continued reliance on direct discharges into our marine waters and combined sewer overflows.

I hope today's witnesses can shed some light on the conflicting interpretations of the data and studies conducted into the dolphin mortality. Mr. Chairman, under your able leadership, I am certain we will be able to settle this debate conclusively. Thank you, Mr. Chairman.

Mr. FOGLIETTA. I thank the gentleman. Before we proceed, I do want to announce that the Subcommittee has received a statement from our distinguished Chairman, Mr. Jones, written testimony from Associate Professor Joseph E. Cummins of the University of Western Ontario, as well as testimony from Greenpeace.

Because of time constraints today, however, we could not have them present their oral testimony, but, without objection, I will enter these statements into the record. So ordered.

[The statement of Mr. Jones follows:]

PREPARED STATEMENT OF HON. WALTER B. JONES, A U.S. REPRESENTATIVE FROM NORTH CAROLINA, AND CHAIRMAN, COMMITTEE ON MERCHANT MARINE AND FISHERIES

Mr. Chairman, I would like to thank you for holding this important hearing today. During 1987, well over ten times the usual number of dolphin deaths occurred in my home state of North Carolina. These 111 instances clearly point out that something is seriously wrong with the environment in which these high level marine mammals live.

The questions posed for us today are many. Did these deaths occur as a result of man's cavalier attitude toward using the ocean as a dumping ground? Might these deaths have occurred as a result of the devastating red tides which also occurred during that year? What are the relationships between the two?

The issue is a complex one indeed. For years, the Committee on Merchant Marine and Fisheries has been concerned about the state of this planet's oceans. Many times, our warnings have been viewed with the typical "Chicken Little—The Sky Is Falling" disregard. What I fear that these dolphin deaths portend is only the beginning of an accounting for our reckless treatment of the life-giving ocean off our coasts.

I am eager to hear from the witnesses invited here today. They are a distinguished and educated lot and I hope that they can shed some further light on this perplexing matter.

[The statements of Professor Cummins and Greenpeace may be found at end of hearing.]

Mr. FOGLIETTA. The first witness this afternoon is Dr. William Evans, Undersecretary of Commerce for Oceans and Atmosphere, and Administrator of the National Oceanic and Atmospheric Administration. Dr. Evans.

STATEMENT OF WILLIAM E. EVANS, PH.D., UNDERSECRETARY FOR OCEANS AND ATMOSPHERE/ADMINISTRATOR, NATIONAL OCEANIC AND ATMOSPHERIC ADMINISTRATION, DEPARTMENT OF COMMERCE

Dr. EVANS. Mr. Chairman, and Members of the Subcommittee, thank you for the opportunity to be here today to review our understanding of the events surrounding the deaths and stranding of the Atlantic bottlenose dolphins on the East Coast during 1988 and 1989. In addition to that, let me say that I appreciate the opportunity to also hear the very deep concerns of the Members of this Subcommittee. That is very valuable to me, in making my evaluation of exactly what the meaning of the report is, so that I can pass this information on to the Secretary of Commerce, who happens to be very interested in this and other issues that have to do with the environment.

We began monitoring the situation, that you discussed with great accuracy, Mr. Chairman, very closely, as did the Marine Mammal Commission, and many other organizations. During the second week of August, the Commission convened a special clinical investigation team which included the Smithsonian Institution, the Environmental Protection Agency, the Animal and Plant Health Inspection Service, the Department of Agriculture, and a lot of people from volunteer groups.

Clinical investigations were considered necessary because in both the Virginia Beach and the New Jersey areas, large numbers of animals washing ashore were obviously seriously ill, and dying as they came on the beach.

The leader selected for the team was Dr. Joseph Geraci of the University of Guelph, a well-known marine mammal veterinarian.

The largest number of strandings were occurring in Virginia Beach, and so the team began its necropsy work in the laboratory facilities provided by the U.S. Navy's Little Creek Amphibious Base.

What we saw happen was an unprecedented die-off of Atlantic bottlenose dolphins that began in the Delmarva area in the early summer of 1987, and progressed on, in time, through the summer, fall, and winter, gradually moving southward.

It became obvious to us, as this was developing, that we would have to spend some effort analyzing the experiences of the response team, and the tremendous amount of data that came out of their activities.

Along with the Marine Mammal Commission and the Office of Naval Research, NOAA developed a plan for funding this follow-up work. Because of his reputation, his familiarity with the events, and the quality of the leadership he had shown on the response team, we contracted with Dr. Geraci to oversee the follow-up studies and prepare a report on these events, and what might have caused them.

We received Dr. Geraci's final report two weeks ago. His report, which he will discuss with you, summarizes the events from the early summer of 1987 to the early spring of 1988, outlines the methodology for conducting the studies, details the findings of the studies, and discusses their implications.

The report concludes that there is evidence that the dolphin mortalities may have been caused by brevetoxin, a neurotoxin arising from red tide, moving up the food chain. Brevetoxin acts directly upon the respiratory system, and has been linked to fish die-offs and respiratory dysfunction in swimmers.

To say the least, this hypothesis has caused quite a controversy even before the report was released. A draft report was circulated to peer scientists for review and comment. Dr. Geraci has considered those comments. Yet, as he will point out, he remains convinced of the validity of his observations and conclusions.

We, in NOAA, accept the report for what it is—the best judgment of our consultant, interpreting available data and looking for a conclusion that best fits the available information. Because of our respect for Dr. Geraci and the response team, we are confident that the analyses were done competently. The conclusions of the report present us with a challenge to investigate new possibilities in cases of marine mammal strandings, and particularly those involving Atlantic bottlenose dolphins.

Brevetoxin poisoning, frankly, was not considered to be a cause of marine mammal strandings and deaths in the past. It is not something we have looked for in these cases, and it may have been a cause that we simply missed in previous strandings, or possibly contributed to some of the strandings we have seen in the past.

I would also note that the report does not rule out other contributing factors, and we need to be aware of these as we plan our future research and monitoring activities.

Many have wondered why ocean pollution was not treated more significantly as a potential cause. I will let Dr. Geraci comment, in detail, but let me also say that I have a great deal of respect for his judgment, that while we cannot rule out the contributing influence of the high contaminant levels, we cannot, based on the available data, tie them to the dolphin mortalities.

And as an aside, Mr. Chairman, I would like to mention that in my own personal experience over the past 30 years—and I have handled a number of beached dolphins—almost every dolphin of which I have taken samples, either of body organs or of blubber, unfortunately, we have found high levels of chlorinated hydrocarbons. This is not an uncommon finding in dolphins. It is not a pleasant one, but it is there, and which I think is a reflection of the fact that we do have a serious problem.

We have a lot of pollutants in our coastal waters. I do not think there is anybody on this Subcommittee, anybody in this room, who questions that fact. Let me reemphasize NOAA's concern for the health of the marine environment. The report recognizes the need to investigate the dynamics of this epidemic, and further, I would certainly support this need.

Mr. Chairman, this was a unique event, both perplexing and alarming. The response team has given us a detailed report of their investigation and findings, including a conclusion as to how this event may have happened.

I would like to add, just briefly, that all of the samples that were collected are being maintained at the Armed Forces Institute of Pathology, that serves as a national repository and makes material available to responsible researchers. We think it is entirely appropriate for any interested group, or agency, to make their own examinations of these materials and come to their own conclusions.

This would be very useful to us, also. Thank you very much, Mr. Chairman, Members of the Subcommittee. I would be willing to try to address any questions you may have. Thank you.

[The statement of Dr. Evans may be found at end of hearing.]

Mr. FOGLIETTA. I thank you, Dr. Evans, and I would like to announce that we will go into the five-minute rule for questioning. I think with all the Members we have here, and all of whom have questions—I think it would be a lot more expeditious to do it in that fashion. So, if we can find a timer somewhere, we will proceed with that. So, being the Chairman, I will proceed without the timer, to start.

Doctor, can you outline for us the peer review process under which Dr. Geraci's report was circulated for review and comment.

Dr. EVANS. Yes, Mr. Chairman. The peer review process that we chose is one that is used by the National Science Foundation. It is a confidential process for scientific review of findings like this. Because of the complexity of this particular piece of research, and the fact that it started out, initially, in one direction, but since we found that it was very complicated, and there were a number of factors involved, we went into a different kind of study.

We thought it was necessary to use the National Science Foundation method. This report was subject to review both inside and outside of NOAA. To ensure the integrity of the process, we needed to keep the identity of the individual reviewers confidential, which is standard for National Science Foundation. The reviewers felt that brevetoxin present presented one reasonable hypothesis, but that some other, as yet unidentified, infectious disease, might also be a cause.

No reviewer of the report hypothesized that the pollutants caused the death, though some noted that they may have contributed. Dr. Geraci took account of the peer review, modified some of his conclusions, but he still believes that the circumstantial evidence points to brevetoxin as the cause.

Mr. FOGLIETTA. Doctor, I understand the need for keeping the names of the reviewers confidential, but is it possible that we can have the unedited comments made available without attribution?

Dr. EVANS. We will make those available for the record, Mr. Chairman.

Mr. FOGLIETTA. I thank you.

[As of August 30, the information from Dr. Evans was not received.]

Mr. FOGLIETTA. Dr. Evans, has any individual with your agency, or another Federal agency, suggested, hinted, or directed that the possible role of pollution in this epidemic not be pursued at this time?

Dr. EVANS. No, sir.

Mr. FOGLIETTA. Were any restrictions placed on Dr. Geraci in the conduct of his investigation?

Dr. EVANS. No, sir. None at all.

Mr. FOGLIETTA. How about budget restrictions? Were there any budget restrictions placed on his effort?

Dr. EVANS. No, sir. Only when I had to start paying the bills at the end of the year, since this was an unbudgeted project.

Mr. FOGLIETTA. I thank you, Dr. Evans. Congresswoman Schneider.

Miss SCHNEIDER. Thank you. Dr. Evans, welcome, and thank you for your tolerance in spending an entire hour listening to us already testify, and——

Dr. EVANS. That was maybe the most important part for me.

Miss SCHNEIDER. Clearly; clearly. Well, we welcome you, and congratulate you on the job that you have been doing, but I do have a few uncomfortable questions to ask here about the report, particularly what appears to be some incongruity between Dr. Geraci's interim report, which was submitted on May 3rd, 1988, versus the final report which was later submitted in April, 1989.

What the question relates to is what appears to be a complete omission of any discussion of ocean dumping, and the role ocean dumping may have played in putting this study together.

I would like to read you a little quote that came from the interim study. It says, "Exploring the possibility that oceanic pollutants combined with unusual environmental events might be associated with the dolphins' condition, a request was made to the EPA for data relating to the dumping of municipal and industrial wastes at the 12-mile and the 106-mile dump sites. Some information on the 106-mile dump site has been provided." Et cetera. Et cetera.

But the point is, that in the final report, nothing is mentioned about ocean dumping nor is there any mention of the information that was requested by Dr. Geraci. And knowing of the intense interest in Congress, and especially before this Committee, in ocean dumping, don't you think that that is a significant oversight, by not mentioning it in the final report?

Dr. EVANS. Well, Congresswoman Schneider, there obviously is a difference between the preliminary report and the final report. I would like to defer to Dr. Geraci to explain why that is. I did read the interim report. I must be very honest with you. I have not had a chance to read the final report. I have been involved in working on a number of other things, but I will read the final report. I think that if it deems that it is necessary to have an expanded discussion on the relationship between the work that has been done on the 106-mile and the 12-mile dump sites, and if Dr. Geraci wants to expand on that, I do not have any problem with him doing it. It may have been removed on the recommendation of one of the peer reviewers. I was not one of the peer reviewers.

Ms. SCHNEIDER. All right. Well, if you keep that in mind in the review of the final report, I think that that would be of interest to all of us. It seems to be an ingredient that may have been left out in the final tally.

The other thing is I understand that the organochlorine figures indicating high levels of contaminants in the dolphins were available in 1987, and I am curious to know why those figures were not released until April of 1989.

Dr. EVANS. I really could not answer the question. I certainly will have it investigated and answer it for the record.

Miss SCHNEIDER. All right. That, too, would be greatly appreciated.

[As of August 30, the information from Dr. Evans was not received.]

Miss SCHNEIDER. Also, as someone as familiar with marine mammals as you are, I wonder if you could explain how the death of a dolphin with PCB concentrations of 6800 parts per million in its liver cannot be attributed to such high signs of contamination?

Dr. EVANS. Well, everything I would tell you would be speculations; but I will say this—based upon the experience I have, and as you know during the past 3 years I have been a little bit more of a technocrat, bureaucrat, than a scientist—so I have not really kept up with the literature. Having looked at a number of animals, the levels that I saw in the preliminary report were considerably higher in Tursiops, both living and dead on the East Coast, and in some of which I am familiar on the West Coast. I am not too sure what that means, or whether or not you can draw a conclusion from that. It may be where the dolphins live. This is a close, inshore population.

I think that it needs to be understood—and I think Dr. Geraci may want to expand on this—that I have taken samples from both, living dolphins that seem to be doing extremely well, especially in an ocean area, that seemed to have very high levels of chlorinated hydrocarbons in their blubber. They seemed to be able to tolerate this much more so than a lot of other animal systems. Some domestic livestock, actually, if you look at them, will have accumulated standards much higher than that which EPA would accept for human consumption.

I think that those relationships are things we do not understand. I think that they certainly are valid questions, and things that we should be spending some time looking at, so that we understand what those levels actually mean, and whether or not they are indicating problems—I think one of the distinguished Congressmen here made a comment, which I thought was very interesting—is that I think the thing that concerns me, as well as a number of the other scientists, is that we are dealing with top-level predators in the food chain.

Miss SCHNEIDER. Right.

Dr. EVANS. What we are seeing is that these are indicator species. We should be, you should be—this group should be very concerned—just as we as scientists should be, because we are being told something. We do not know what it is. Brevetoxin may very well be a very—and probably is a very-significant portion of this—

but I agree with the Members of the Committee. I do not think it is the entire story.

Miss SCHNEIDER. Thank you.

Mr. FOGLIETTA. I hate to interrupt but I think your time is up.

Miss SCHNEIDER. My time is up; yes. Thank you.

Mr. FOGLIETTA. Thank you. Congressman Pickett.

Mr. PICKETT. Thank you, Mr. Chairman. Dr. Evans, were there any State or local, or private organizations that investigated this condition affecting these dolphins?

Dr. EVANS. We had a number of private organizations who were participants in the group. I do not know the names of all of them, specifically, Congressman, but there were others. I am sure that Dr. Geraci can give you a listing, and some of the other witnesses can give you a listing of all of the organizations.

I was absolutely amazed when we put out the call, with limited resources, to start this program, that we had volunteers from private hospitals and private clinics to analyze blood samples, and to do a variety of things. It was an incredible, I think, example of volunteerism, if you will, of people jumping in to address a very severe and a catastrophic event. I was very impressed by that.

Mr. PICKETT. Did any of these organizations, or individuals arrive at any tentative conclusions about what, in their opinion, was the cause of the deaths?

Dr. EVANS. I am really not aware of what their reports may have been to Dr. Geraci. I am sure he can address that issue, Congressman. I really do not know.

Mr. PICKETT. Is he the only one that is aware of all these different reports that may have been prepared?

Dr. EVANS. He is the person who basically collated all of the information that came in from samples—as he will explain to you—were collected and were sent out in what we call a double-blind study, to a number of different places. In fact there were a lot of control substances that were also used. Tissues from animals that, for instance, were not dolphins, were involved, so there was a very complicated study—I do not really know the experimental design that well, and I would like to defer to him on that, sir.

Mr. PICKETT. Thank you very much, Dr. Evans.

Mr. PALLONE. [presiding.] Thank you. The gentleman from New Jersey. Mr. Saxton.

Mr. SAXTON. Thank you. Dr. Evans, would you care to expand on the notion that you and others have talked about, that brevetoxin may have been a primary cause, but there may have been other elements that were contributing factors, or perhaps major causes as well.

Can you give us a better idea, in terms of——

Dr. EVANS. Well, what I am doing, Congressman Saxton, is reporting to you, based on my knowledge—which is the same as yours—the preliminary report, and a very quick reading of the other report, in terms of what Dr. Geraci's concerns were.

The study that he had indicated that although—you showed the small sample sizes there—I think what I mentioned earlier—the fact that most of the animals that we looked at seemed to have certain levels of chlorinated hydrocarbons in the blubber and the liver, and other samples.

I think that as we got down to look at the brevetoxin, it is my understanding that the sample size did get smaller. They are looking at very specific pieces of tissue. I do not really remember all of the details of what was done on the study, except that the fish in the stomachs, and some of the other things were isolated as having amounts of brevetoxin.

Brevetoxin we know is a toxin, and it can cause death, and I think that may be in one of the things describing the assumptions that Dr. Geraci is making.

Mr. SAXTON. Thank you. Dr. Evans, do you have any indication, or knowledge as to when the conclusions may have been drawn that brevetoxins were to be considered the primary cause, and other agents were not?

Dr. EVANS. The first time I was made aware of Dr. Geraci's conclusions was at the press conference, which we actually had prior to having the report reviewed. Because of the importance of this issue, because of the public concern about it, we felt that it was important even though the information was preliminary at that time, and had not yet gone through peer review, to give Dr. Geraci the opportunity to state his conclusions based upon the information that he had.

That was the first time I became aware that brevetoxin was the material. I think this is something that happened towards the latter part of the study and the tests, and I think that was related to the fact that we did have the mass stranding of humpback whales, that were associated with having consumed large amounts of mackerel which apparently had toxins associated with them.

Mr. SAXTON. Well, I would like you to react to this. I have before me a memo which I received this morning. It is from an individual in NMFS, and is dated September 9th, 1987.

And it is quoting a person who apparently was taking part in the study. And this person requested a copy of data generated on PCBs and pesticides for his own personal use.

And in September of 1987, it says, he indicated that, "No special attention will be drawn relative to this data, and that a blanket statement will be made, that the levels of these components are not out of the ordinary."

Now that was September of 1987, according to this memo that I have in front of me. Does that seem reasonable to you?

Dr. EVANS. It does not seem reasonable to me, but I would like to know more about it and would like to investigate it.

Mr. SAXTON. Well, perhaps we should talk about this privately, and if we decide that it should be something pursued publicly, we can pursue it that way.

Dr. EVANS. I would be more than willing to do that, sir.

Mr. SAXTON. Thank you. Thank you, Mr. Chairman.

Mr. FOGLIETTA. [presiding.] Congressman Pallone.

Mr. PALLONE. Thank you, Mr. Chairman. Dr. Evans, in your testimony, you acknowledged that the brevetoxin hypothesis caused quite a controversy even before the final report was released. Isn't it fair to say that NOAA's highly unusual approach of announcing its conclusion nearly three months before releasing its final report contributed to this air of controversy and skepticism?

Dr. EVANS. Congressman, in all fairness, first of all, the press conference we had was not set out as giving a definitive finding. It was clearly stated in the press conference that these were preliminary results. It was clearly stated by me, in my introduction at the press conference, that we were releasing this information because of its importance to people, because of a lot of other things, to try to dispel the fact that maybe it was not brevetoxin, maybe it was not chlorinated hydrocarbons, but it sure as heck was not AIDS. There were a lot of people that were concerned about health. It was having an effect on beaches. Although we had preliminary information that had not even been peer reviewed by people within NOAA, it was my value judgment to allow Dr. Geraci to go through and mention the report, and say what he had done up to that point and what the results were. I think it was an important thing to do. I would do it again.

I think that maybe I would stress, even more, it was preliminary information, and I think that the reason it became controversial after the press conference is some of the conclusions Dr. Geraci came to were different some others who thought it might have come from—high levels of chlorinated hydrocarbons in various animals.

I think it was important to get the information out, but it was preliminary, it was stated as preliminary, and in fact you even mentioned the delay. The delay in your getting the report is because we sent it out to members of the Marine Mammal Commission Committee of Scientific Advisers, to people in the academic community, as well as people internally within NOAA, in order to review it and review all of the data. That was one of the reasons there was a holdup in the report. It takes a fair amount of time for everybody to write back. I think if you check with the National Science Foundation, you will find that it frequently takes several months for a peer review of a scientific paper.

Mr. PALLONE. I did not attend the press conference but I saw the press release, and of course the press coverage afterward, and I got the distinct impression they were saying that "red tide" was the cause.

The last sentence in the report, in the discussion, says: "Equally important is the need to resolve the growing question of whether contaminants at levels found in the dolphins might have affected their resilience, and rendered them more susceptible either to the toxin or to the microorganisms that eventually brought them to their demise."

When I read that last sentence, Doctor, I got the impression that basically the report did not carry out the mandate of the law.

Congressman Carper mentioned that his amendment specifically requires examinations of certain aspects of the dolphin epidemic, and specifically includes the extent to which pollution may have contributed to the epidemic.

When I read this last paragraph, which lays open the whole question of whether pollutants really caused, or were a major contributing factor to the fact that the dolphins were no longer immune to the brevetoxin and to the "red tide", I have to conclude that the provisions of the Carper Amendment were simply not met.

My question to you is: Do you believe they have been met? You seem to indicate that you would be in favor of re-opening this investigation to look at the extent to which chemical pollutants may have been a contributing factor.

I would like to know whether or not you would be in favor of reopening the investigation to look at those pollutants as a contributing factor, because I think it should be opened again.

Dr. EVANS. Well, Congressman Pallone, you are entering an area which is budgetary. You know, in terms of having other people look at the materials, it is all now available. It was not available at the time the study was going on because of the nature of the kind of study that was being done. All samples are now available.

Anybody who wants to look at it—and I would encourage organizations, universities, and others—or who would like to take this material and run their own test, to please do so. I think it would be very useful to us.

But at the present time, we are concentrating on the other phase of it now. We have a very serious problem here. We have 750 dolphins that died. We are still in the process of trying to determine what impact that had on the population. That is one of the things we are supposed to be doing, according to the changes that Congress has made, and the tightening, and I think improvements that have been made in the Marine Mammal Protection Act.

We are trying to address that issue, and to look at what the effect has been on the population. We have increased our efforts in looking at what the impact is on the number of animals in the Atlantic Coastal stock *Yursiops*. We are also looking into the Gulf of Mexico population. There is an enigma in the Gulf of Mexico—red tide and no dead dolphins.

Mr. FOGLIETTA. I thank the gentleman. Mrs. Saiki.

Mrs. SAIKI. Yes. Thank you, again, Mr. Chairman, the indulgence of you and the rest of the Committee Members, because I am going to move you over from the East Coast all the way over to the Pacific.

Dr. Evans, just in general, all over the United States, do you know how many dolphin deaths there have been, of dolphins kept in resort pools?

Dr. EVANS. All over the United States?

Mrs. SAIKI. Yes.

Dr. EVANS. A couple of years ago, I used to know, but I really do not know right now. I know that there are quite a few in Florida and California, and Hawaii has a number. My last recollection was maybe three or four places where they were being kept. By "resort pools," you are talking about where people go to visit? Not display? Not public display?

Mrs. SAIKI. Display as well.

Dr. EVANS. Oh, public display?

Mrs. SAIKI. When you have dolphins in pools, contained, man-made pools, whether they be in a hotel, or whether it is at a roadside stand or——

Dr. EVANS. No. I really do not know the exact number.

Mrs. SAIKI. Does the agency intend to get some numbers on this?

Dr. EVANS. The agency has very accurate numbers of the animals that are in public display.

Mrs. SAIKI. Well, would it be possible for the Committee to get those numbers?

Dr. EVANS. We certainly will provide it for the record.

[As of August 30, the information from Dr. Evans was not received.]

Mrs. SAIKI. Could the Committee also get the information as to how these dolphins died; what caused the deaths.

Dr. EVANS. There are necropsy reports which are available on all dolphin deaths, and if you would like—there have been a lot of dolphins in captivity over the years, and there are probably a lot of necropsy reports, but those certainly would be available for the Committee to look at, if they so desire.

[As of August 30, the information from Dr. Evans was not received.]

Mrs. SAIKI. In monitoring these deaths, is there any similarity of the causes of these deaths?

Dr. EVANS. Similarity in what way, Congresswoman?

Mrs. SAIKI. Well, if you are going to keep dolphins contained within an area, if there is a similarity as to the causes of these dolphin deaths, perhaps we could get information together to prevent them in the future.

Dr. EVANS. Well, some of the dolphins in captivity die of old age. Most of them have complications with pneumonia, and I think you will find that Dr. Geraci will mention—you might want to ask him that question, too, since he is a veterinarian for a number of the organizations that maintain animals in captivity. But respiratory diseases, of one sort or another, are quite a common secondary cause.

Animals have died in captivity of a variety of things, including several different kinds of diseases, kidney failure, and even coronary problems.

Mrs. SAIKI. Well, I would be more interested as to whether pollutants have anything to do with it; whether circulation of water; the diseases that are prevalent; and also, in the Swim With A Dolphin program, whether the introduction of the human person with these dolphins cause any problems at all. Because I noticed that throughout the country there are more and more instances where people are trying to promote their hotels by allowing people to swim with their dolphins.

I would just like to get this information, if it is possible.

[As of August 30, the information from Dr. Evans was not received.]

Dr. EVANS. In reference to the Swim With A Dolphin program, I would like to say, both, for you and for the record, that as the Chairman of the Marine Mammal Commission, as the former Director of the National Marine Fisheries Service, and now, in my current position, I have been very concerned about Swim With Dolphin programs, and the implications. We have tried to put a number of regulations into place to make sure that the animals are tested to determine that they do not have diseases that could be passed on to the humans, and of course we have another problem. Dolphins, too, can catch human diseases, and so we have a concern about that.

In particular, there is presently a situation in Hawaii where, at one of the major hotels, some dolphins have recently died. There is some indication, I believe in that case, that they may have eaten fish which had toxin. I know, being from Hawaii, you are quite familiar with saxitoxin. It is a major problem in tropical areas. We are looking at that very carefully, and we are going to be evaluating that whole situation, and until such time as we have some concerns—and we know that there was nothing involved that could cause human health problems, we are going to monitor that situation very closely.

I, personally, am very concerned about that, and will personally be looking at that program.

Mrs. SAIKI. Well, I, personally, would appreciate it, and look forward to whatever report you can give to this Committee so that we can watch this thing. Thank you.

Mr. FOGLIETTA. Congressman Hughes.

Mr. HUGHES. Thank you, Mr. Chairman, and thank you, Dr. Evans. Dr. Evans, getting back to the question that was asked by my colleague, Mr. Pallone, it would seem rather clear, that the last sentence dealing with contaminant levels found in these dolphins raises the question as to whether you have really answered the question directed by the Congress.

The Carper Amendment had five specific areas. The cause or causes of the epidemic, which is questionable to this point. It is very controversial.

The effect of the epidemic on the coastal and offshore populations, which we have not yet addressed, and you have just indicated needs to be addressed.

The extent to which pollution may have contributed to the epidemic, which has not been addressed. Whether other species and populations of marine mammals were affected by those factors has not been addressed.

And any other matters pertaining to the causes in fact of the epidemic.

Now I hear what you say about it being a budgetary matter. I do not consider it a budgetary matter. The law provides for you to address those issues, and——

Dr. EVANS. Congressman, I think we did address those issues in—you know—and I think that Dr. Geraci's report may—you may feel that there are some shortcomings in it, we will certainly look at those, but——

Mr. HUGHES. Well, he says, "Equally important is the need to resolve the growing question of whether contaminant levels found in dolphins might have affected their resilience and render them more susceptible"——

Dr. EVANS. But the data that he had at the present time does not indicate—according to him—that that was the cause at this time. That he is still supporting, and still believes, that brevetoxin was the major cause of deaths in this particular case.

Mr. HUGHES. Well, I am not a scientist, but I have seen algae blooms and red tides for decades, particularly down in the Gulf Coast, but also off of our coast. We have never had a dolphin mortality anywhere comparable to that. Now I am not a scientist, once again, and I see, test results that are based upon testing something like 17 dolphins and getting eight indications that, in fact, there is some indication that brevetoxin is present in eight of those. And yet, in all 80 of the dolphins that were sampled, there were high levels of contamination.

I do not understand how, as a scientist, you can ignore the high level of contamination. What is even more troubling is that the only thing that apparently was tested for were the usual toxic organic substances, such as PCPs, DDT, and DDE. We did not test for any other toxic chemical substances, and I do not know how, as a process, we can determine just what caused their deaths, unless we look at the total picture. Can you explain to me how——

Dr. EVANS. Well, I would suggest that you ask Dr. Geraci, and if he cannot give you an adequate answer——

Mr. HUGHES. Well, we will.

Dr. EVANS. ——that is something we need to address.

Mr. HUGHES. We will, but I am asking you, because you happen to be the first witness here, and, you are in charge of the agency that basically has to carry out the study. As a scientist, does it trouble you, that we did not test for other organic substances, and that we have taken the conclusions based upon samplings in eight dolphins out of 17 that tested positive for brevetoxin, and we basically have dismissed, almost out of hand, the fact that in all 80 of the samples we found that they tested positive for toxic substances, high levels of toxic substances?

Dr. EVANS. As I mentioned earlier, Congressman, I think that you will find that it is—I did not say it was acceptable. It is relatively common to find chlorinated hydrocarbons in the livers and in the blubber of marine mammals. That does not make it right. It just is a common thing.

Mr. HUGHES. The levels that were found——

Dr. EVANS. Some of these levels were not necessarily high enough to cause a major concern.

Mr. HUGHES. But the point is, not in the levels that were found in these samples. My question was—and you have not answered it—does that trouble you, that we apparently just dismissed that out of hand, when in fact the mandate of the Carper Amendment was to determine the extent to which pollution may have contributed to the epidemic?

Dr. EVANS. Congressman, when I read the final report, I will write you an answer for the record. I have not read the final report.

Mr. SAXTON. Will the gentleman yield.

Dr. EVANS. I have read the preliminary report.

Mr. HUGHES. I will be happy to yield to the gentleman.

Mr. SAXTON. Dr. Evans, this is the point that I was trying to make in referring to the memo that I referred to, and I will not be specific about the memo because I just received it this morning, and have not had a chance to—I do not want to take it out of context. But this memo very clearly states, that in September of 1987, a decision was made by at least one person who was working on this project, who was privy to meetings that are referring to in this memo, that, again, quote, "No special attention will be drawn relative to this data, and that a blanket statement will be made that the levels of these components were not out of the ordinary."

Dr. EVANS. Well, that is not good science, and that is certainly not what we want, Congressman Saxton. That is not what I want. When I find out more about where that memo came from, if that is indeed a true statement and somebody said that, then I will investigate it and we will fix it, and we will open this whole thing up and start the whole darn thing all over again.

But, you know, that is the point. You are giving me something out of context that I am not familiar with, and we do not know who wrote it.

Mr. SAXTON. Well, I apologize for that, and I was tempted to make this memo public, but I decided that I would not do that until we have a chance to examine it, and you have a chance to look at it and decide whether there is an explanation for it. But the language in it seems——

Dr. EVANS. Well, no, there should be no explanation for statements like that.

Mr. SAXTON. The language in it seems very——

Dr. EVANS. That is quoting the data one way or another, and that is the main reason that we went along with the approach that Dr. Geraci wanted to use, is to make sure that we stayed away from that, and got what we felt was an unbiased type of answer that addresses the things that are laid out in Congressman Carper's bill.

Mr. SAXTON. I have to say to you, sir, that I am not sure that these were meetings—the meetings that are referred to in this memo had anything to do with Dr. Geraci directly, but they do appear to have to do with the National Marine Fisheries people, and meetings that were being held relative to this subject.

Perhaps what we should do is to let you have a copy of this, and examine it, and——

Dr. EVANS. I would appreciate that very much because I would like to investigate it, and I will report back to this Committee on it.

[As of August 30, the information from Dr. Evans was not received.]

Mr. SAXTON. Thank you very much. I thank you, Mr. Chairman.

Mr. FOGLIETTA. I thank the gentleman. Congressman Goss.

Mr. GOSS. Thank you, Mr. Chairman. I, too, would like to echo my colleague's sentiments about the Swim With A Dolphin program information. If that could be made available, it would be helpful.

I think that you mentioned that the "enigma" is in the Gulf of Mexico. I would take exception to that. I think the enigma is in Dr. Geraci's report, and I say that from the perspective of someone who lives on the shores of the Gulf of Mexico, and have lived for 20 years, and watched red tide come and go, and the dolphins seem to get along well, and I am certainly the first to admit that there is a potential that brevetoxin is considerably more potent elsewhere because of other combinations of factors.

But accepting the report that we have got before us, and addressing it, I think we are being asked, or it is being suggested that we sort of shrug this off as a natural event, and if this is a natural event, if we have lost 50 percent of the near-shore dolphin population this year, then perhaps we cannot expect to see many more dolphins around if we have the misfortune of having another red tide in another year.

I do not think that really is a conclusion that anybody is ready to accept right now—I am certainly not—without a lot more information. And my reasoning is very simple, and my question to you is rather simple.

In your estimation, do we know enough about red tide, and, in your estimation, do we know enough about the effects of red tide on dolphins? Both of those questions, it would seem to me, cannot be answered within a year, but it seems to me that is what we are being asked to accept.

Dr. EVANS. As a scientist, my answer to that question would be no, I do not think we do.

Mr. GOSS. I think that is a fair statement. So, as an administrator, what do you suggest the recourse should be?

Dr. EVANS. Well, I think that, again, it is something that, as we are looking at it—and I think Dr. Cross is here from one of our laboratories, who has been specializing in that area, and I think he may address that—but I think that we need to continue to investigate not only the effects of red tide in dolphins but the effects of red tide in seafood in general.

And I think the whale issue raised a major thing. I think we need to know more about the interactions between this and marine mammals. Going back to Hawaii again, there has been some speculation—but we still do not know—that the rather drastic decrease in monk seals, in Hawaii, which is—this is probably the most endangered marine mammal around right now—and that some of that may actually be due to the fact that these animals are found in and around reefs.

And it has been speculated that maybe because they have been eating various reef fish, that part of the weakening of them, or the cause of death, or other sorts of things that we do not really understand, have been caused by ingestion of saxitoxin, and maybe some other kinds of toxins that we do not know. We just do not know a lot of these things. I think it is important—and you raise an important point. I think that probably the most important thing that has come out of this disaster is that it is causing us to ask a lot of questions that we never asked in the past.

We have had mass strandings in the past. We never looked for toxins. We will now.

Mr. GOSS. Well, I think that our desire here is to help you do the job, that we would like to see done, so we all know more, and I certainly feel that with the competition we have for dollars this year, we have to make the case. I think nature has made the case for us. The question is, where do we go with it from here, and I just want to determine that there is a willingness to proceed, if we provide the resources, and I hope there is.

Dr. EVANS. This is something that I think we need to look at very closely.

Mr. GOSS. Thank you, sir. Thank you, Mr. Chairman.

Mr. FOGLIETTA. Congressman Carper. I thank the gentleman. Congressman Carper, please.

Mr. CARPER. Thank you, Mr. Chairman. Dr. Evans, welcome. We thank you for your presence today, and for your testimony.

Mr. Pallone has already said it; Mr. Hughes has echoed it; Mr. Saxton has again reemphasized it. And I do not want to beat a dead horse. But we are not asking you to start all over, Dr. Evans. I think what each of us are asking you to do is simply to ensure the job that has been begun is finished, in the spirit that we have asked you to finish it.

Every Member, every Member of this Committee voted last year in support of an amendment that did the five things that Mr. Hughes has alluded to, and a third of those was to determine the extent to which pollution may have contributed to the epidemic.

Now, you may be satisfied, in your own mind, that the preliminary study concluded thus far addresses that issue. You have some doubters over on this side of the table. The question I would ask of you, sir, is, what further steps do you intend to take to ensure that the spirit, and the letter of the law—of the law—are complied with?

Dr. EVANS. First of all, Congressman, I am going to become much more familiar with your law than I was when I sat down at this table. That is the first thing I am going to do.

The second thing: I am going to sit down with all my staff and go through each one of the points that Congressman Hughes and Congressman Pallone looked at, and get an evaluation of where we are on that. If we have not been there, then we are going to respond to you, in writing, where we think we are and what we are going to do to remedy that.

I mean, the intent is there, and we certainly want to go with the intent. I have to admit that I am not as familiar with that piece of legislation as I should be. I have some staff that maybe should have made me a little bit more aware of it than they have, and they will, shortly after we have this hearing, make me a lot smarter on where we have been with that than I am at the present time.

But I understand your concerns. I hear the concerns of this Subcommittee and I will respond to them.

Mr. CARPER. Thank you. I appreciate very much that statement. In the amendment that we adopted, we directed the agency to coordinate with a host of other agencies in reaching the conclusions that we had requested. Among the agencies that we expected you to consult with and to cooperate with was EPA.

Let me just say: Do you anticipate, or have you come into any problems in that relationship with other agencies, particularly with EPA, in looking at the points raised within this issue, raised by this report?

Dr. EVANS. As far as I am aware, we have not. In this particular issue, again, I am not aware of whether or not there has been any problems. Our relationship with EPA, in a number of other issues having to do with everything from the mussel watch to the Status and Trends program, and a number of other programs that we have, has been very good, and we are in the process, now, of working on things like acid rain and a number of other things, in a cooperative spirit with EPA.

So I do not know why we would be having any problem with EPA, or any of the other agencies, because we have a number of memoranda of understanding, we are entering into a whole new, very good relationship with those organizations, doing a lot of cooperative work.

Mr. CARPER. I am going to ask you, if you would, to respond, just for the record, to the extent the cooperation, or lack thereof, that you have experienced from the other agencies that were directed, in our legislation, to cooperate with you and your agency.

Dr. EVANS. We will provide that for the record, sir.

[As of August 30, the information from Dr. Evans was not received.]

Mr. CARPER. Thank you very much. One last question. One last question, for the record, if I could, Mr. Chairman. May I?

Mr. FOGLIETTA. Without objection.

Mr. CARPER. Was this report reviewed by OMB?

Dr. EVANS. Was the report reviewed by OMB?

[Laughter.]

Dr. EVANS. I do not know any report we have that has not been reviewed by OMB, but this report was not reviewed by OMB.

Mr. CARPER. Fair enough. Thank you.

Mr. FOGLIETTA. Congressman Tallon.

Mr. TALLON. Mr. Chairman, thank you, and Dr. Evans, thank you. We appreciate you being here, and your willingness to cooperate, and I think you probably understand that on this panel there are many of us who just cannot accept, with what we have seen, that this is a natural occurrence. I am one that is very concerned about our marine habitat, and our fisheries industry, a shell fisheries industry in my State that is constantly opened and closed because of pollution, and pollution concerns, and an avid offshore fisherman.

But also, we have a highly evolved marine mammal that is showing up—that is the 55 on South Carolina beaches—and another concern that goes beyond the environmental concerns. The largest industry in my State is the $4.2 billion tourism industry, and those beaches are the centerpiece of that tourism industry.

This concerns me very much, and it concerns a lot of our visitors that are coming to enjoy the most beautiful beaches in the world.

Dr. Evans, is there a proposal, or a plan in development for examining the pollution question further? I think you said something about, or suggested that in your statement, and I think Dr. Geraci also urged that.

Dr. EVANS. There is a $12.4 million initiative called the Coastal Ocean Initiative in NOAA's 1990 budget, which breaks out into a number of issues which mostly are pointed towards trying to get better definition of some of the causes and the mechanisms involved in, particularly, coastal ocean pollution. We are certainly going to look at the offshore areas, but as the National Ocean Studies Board, National Academy of Sciences, has said in the past, oceanography and a lot of the marine chemistry we have done has concentrated on deep-water oceanography.

Now is the time we have to understand our bays and our estuaries, and our coastal waters better than we have before. We have gotten some very strong signs from nature, that we had better start doing our homework in this area, and that was the reason we put this initiative in. It is in the budget. It has been presented to both the House and the Senate Appropriations.

Mr. TALLON. Thank you, sir. That is vitally important. I think that will help us and it is certainly a direction we need to move in. I do not have any more questions, Mr. Chairman.

Mr. SAXTON. Mr. Chairman, may I ask unanimous consent to proceed out of order for one minute.

Mr. FOGLIETTA. Yes. Without objection. The gentleman may proceed.

Mr. SAXTON. Dr. Evans, this is kind of a general question, and yet it is specific—and again, I am not a scientist—but have we specified, or has NOAA specified, or is there specification as to what constitutes a specific level of PCBs or brevetoxin? And I ask that, and I will put it in this context: it is kind of confusing, to me, to look at the results of diagnosis of dolphin liver samples that were taken, or analyses of dolphin liver samples that were taken.

In one case, the report seems to indicate that there were 93 nanograms per gram found. In another case, there were 15,820 nanograms per gram found, and, in another case, 310. And those seem like they are numbers that are kind of all over the ball park. Do we have a standard to which to compare——

Dr. EVANS. Well, I certainly would have to ask Dr. Geraci that because that is not my area of expertise, but what were the weights of the animals that were involved? I think that is probably—the size of the animal may be having something to do with what those weights are. But if those are all animals of the same weight, yes, that is all over the ball park.

If it is a young calf, or a very old animal, probably not so. But in terms of the standards, again, my expertise is involved with them when they are alive, or in such a state of death that you really could not be able to tell too much about them because I work mostly with skeletal material and with live animals. So the physiological and the pathology area of it, I am not really knowledgeable. I would suggest you——

Mr. SAXTON. As far as your knowledge takes you, then there is no kind of standard that these numbers can be compared to?

Dr. EVANS. There may very well be, but I am not familiar with it.

Mr. SAXTON. Thank you, Mr. Chairman.

Mr. FOGLIETTA. I thank the gentleman. I have no further questions for Dr. Evans. Do any of the other Members of the Committee have any questions?

Miss SCHNEIDER. No, Mr. Chairman.

Mr. FOGLIETTA. Dr. Evans, we thank you for your testimony.

Dr. EVANS. Thank you very much, Mr. Chairman, and Members of the Subcommittee. Thanks very much.

Mr. FOGLIETTA. Our next panel is Dr. Theodore Smayda, Dr. Pierre Beland, Dr. Gabriel Vargo, Dr. Melvin Goodwin, Dr. Daniel Martineau, and Dr. Harry Smith.

Welcome, gentlemen. This is a very distinguished panel. This is a very complex subject. I would ask that you try to limit your oral presentations to five minutes, if possible, so that we can leave ample opportunity for questions.

With that, I would like to ask Dr. Smayda if he would testify first, at the request of our Ranking Minority Member.

STATEMENT OF THEODORE J. SMAYDA, PH.D., PROFESSOR OF OCEANOGRAPHY, GRADUATE SCHOOL OF OCEANOGRAPHY, UNIVERSITY OF RHODE ISLAND, NARRAGANSETT, RHODE ISLAND

Dr. SMAYDA. Mr. Chairman, and Members of the Subcommittee, I wish to thank you for this opportunity to make this presentation in connection with today's hearings.

I also wish to acknowledge, and extend my appreciation to you for your interest and concern, and sensitivity about such a matter, because I think in the future it will be increasing, and we will need greater congressional interest and help to resolve some of the key scientific problems.

My specialty within biological oceanography is to study the marine phytoplankton. These are microscopic algae which have existed for more than 3 billion years. They are the basis of the foodweb in the ocean. They oxygenate the waters, and through photosynthesis they grow, and they enter into the foodweb. Phytoplankton are eaten by zooplankton and they work their way up to fishes. They are fundamental biogeochemically, in the long term, as well as presently in the world's oceans.

Within this group we have certain organisms that periodically enter into "red tides." These are organisms that have particularly a very potent natural toxin, and for reasons that are still obscure, they proliferate wildly, and, in the process, they transfer toxins of various sorts within the foodweb. For example, herring in the Gulf of Maine oftentimes die off during poisoning because of red tide blooms.

We have paralytic shellfish poisoning. Human deaths occur because of eating shellfish that have ingested toxins produced by certain phytoplankters, and so on. It was therefore with considerable interest that I read Dr. Geraci's report, that the dolphin die-off was most probably attributable to the occurrence of brevetoxin.

After carefully reading this particular document, I have come to the conclusion that, at best, this conclusion is tenuous, and neither convincing nor conclusive.

I base this, I must emphasize, from the vantage point of my experience as a phytoplankton ecologist in foodweb dynamics, and not as a veterinarian, which I am not, nor a pharmacologist, nor a toxicologist, nor a pathologist.

There are several problems associated with this, and, fundamentally, it boils down to the following. The source of the toxin, and how do you get it into the foodweb, so that you can have dolphins dying off on the coasts of New Jersey, and Maryland, and Virginia?

I must emphasize that there are no known brevetoxin producers amongst the phytoplankton, that are indigenous to the Atlantic coastal waters. The toxin-producing organisms simply are not present, ordinarily. And so our problem then becomes, where does it occur?

It is interesting that, in fact, in October of 1987, there was an introduction of Ptychodiscus brevis most probably transported from the Gulf of Mexico, off Cape Hatteras, of the order of 20 million cells per liter which is a prodigious number.

I wish to remind you, though, that there are more than 150 dolphin deaths that occurred off the New Jersey and Maryland coasts long in advance of this particular outbreak of the brevetoxin producer.

You might say, well, okay, there was transport through migration. The best information that I have been able to get indicates that the dolphins, or bottlenose dolphin, already begins its migration in early spring. The menhaden that are potential vectors or transporters of this toxin have already completed their migration by spring.

In fact there is a report in the literature which essentially says there is no significant migration of menhaden, either north or south of Cape Hatteras, the site of the Ptychodiscus bloom, after June. Essentially what we have, then, is a problem of how do you get that toxin, long in advance before it showed up, apparently off the Cape Hatteras coast to cause the die-off along the Atlantic coasts of Maryland, Virginia and New Jersey?

The dolphins cannot ingest Ptychodiscus directly. It is not a substance that is liberated into the water column. They must get it through the foodweb. And if you go through the various scenarios, you quickly find out that there is considerable uncertainty as to the migration patterns.

Dr. Kenney of the University of Rhode Island has come up with the notion that there is no convincing evidence that, for example, the stock south of Cape Hatteras commingled with those stocks north of Cape Hatteras, even though there is migration.

And so to make essentially a long story short, is no matter which point of entry you get into this situation, there is no evidence that one could have a continuous injection of intoxicated fish in the diet, containing Ptychodiscus, along the extended period, and from 1500 kilometers of coastline, to support the notion that there would be deliveries of brevetoxin at the required doses, and at the required frequencies, sufficient to cause this particular die-off.

Other reasons can also be mustered, but I remain skeptical for a lot of reasons that we can go into later, perhaps, that brevetoxin was the responsible lethal factor. There are serious, serious problems with that.

I must commend Dr. Geraci very, very highly, however. Like a lot of us, he was into a crisis scientific management problem, or an exploration problem where things were happening. It assumed political and journalistic features, and I think he did an absolutely marvelous job getting the information that he did do, and getting the people with the requisite skills, and so on.

I do not deny—I do not deny that eight of the dolphins had brevetoxin. It is not surprising that there would be adventitious accumulation of brevetoxin, grazing on the normal part of the foodweb, certainly some, given the presence of this Ptychodiscus in October, there, that you would have this particular entry of a toxin.

But there is a parallel matter of extraordinary consequences occurring in parallel with catastrophic marine biotic events such as the bottlenose dolphin die-off, that I also would like to bring to your attention. It is also relevant to the red tide and nuisance phytoplankton bloom.

An epidemic of nuisance phytoplankton blooms is spreading throughout the sea, accompanied by anoxia, marine mammal, fish and invertebrate die-offs, human deaths and illnesses, and trophic dysfunctions. Regions previously free from toxic phytoplankton blooms now suffer such blooms. Species previously benign have become toxic or nuisances. In many regions——

Mr. SAXTON. Doctor, excuse me. Would you move the microphone just a bit closer, please.

Dr. SMAYDA. Sorry.

Mr. SAXTON. Thank you.

Dr. SMAYDA. In many regions, the frequency and intensity of red tide outbreaks have been increasing. Human deaths due to paralytic shellfish poisoning are increasing. Bloom events are normal aspects of phytoplankton dynamics essential to marine foodwebs, but blooms collectively known as "red tides" represent population explosions of species which are undesirable or toxic to grazers.

A significant global increase in kills of commercially important finfish and shellfish, both natural and cultivated stocks, has accompanied the global surge and spreading of nuisance phytoplankton blooms.

Remarkable die-offs of whales, and perhaps dolphins have recently been linked to toxic blooms for the first time. Enormous financial losses have resulted to commercial fisheries and associated industries, sometimes exceeding $100 million per bloom outbreak.

Marine aquaculture is presently an uninsurable activity because of the highly unpredictable, episodic nature of lethal red tide blooms. Curiously, finfish and shellfish aquaculture activities themselves frequently stimulate red tide outbreaks in the growth area.

Red tide outbreaks are not a new phenomenon. Historical references to such blooms date back to Homer's Iliad and the Odyssey. Episodic red tide blooms are natural events. What is new is their global spreading, increased frequency, and associated catastrophic die-offs of marine animals. Red tide outbreaks are not restricted to dinoflagellates, to Ptychodiscus.

Brown, yellow, white, and green water discolorations accompany bloom events of other phytoplankton groups. What is new is that groups previously considered to be benign now produce inimical blooms. Diatom blooms, for example, have caused fish kills and mussel toxicity, leading to human death, amnesia, and epilepsy.

Red tide blooms, historically, have been primarily colder-water phenomena. What is new is their present proliferation in tropical and sub-tropical waters, accompanied by increased outbreaks in temperate and boreal seas.

The eastern coastal waters of the United States, historically, had been relatively free of toxic red tide outbreaks. What is new is that since 1972, there have been at least six major toxic blooms in the waters stretching from Massachusetts to North Carolina.

In September 1972, New England had its first serious paralytic shellfish poisoning epidemic following a red tide. At least 26 people were poisoned and the clam beds were closed down at a revenue loss of about $1 million per week.

The causative organism has since spread, causing recurrent toxic blooms along much of the New England coast, causing periodic closure of the shellfish areas.

During the summer of 1976, a large anomalous bloom of the dinoflagellate Ceratium tripos occurred in the New York Bight. Ungrazed, its growth eventually became limited by nutrients such a nitrogen, the population sank into bottom waters, rotted, used up the available oxygen and caused anoxia.

Significant mortality of commercially important fishery species such as surf clams, scallops, lobster, and certain finfish ensued. The estimated commercial revenue loss was $64 million.

Since writing that, I have checked this further, and looking into the evidence of Figley, and co-workers, where they evaluated the effect of recruitment of these stocks, and eventually fishing activities, they projected, in 1979, that the economic loss associated with this particular bloom was $569 million.

I have been told that the environmental conditions similar to 1976 are currently found in the New York Bight, and that this region is now being monitored by NMFS scientists.

In summer 1985, an extraordinary brown tide occurred simultaneously in Narragansett Bay, Long Island coastal waters, and Barnegat Bay, a mesoscale event. The causative factors remain unknown. The causative organism was previously unknown to science, even to the genus. Enormous die-offs of mussels and scallops occurred. The Long Island embayments have been particularly impacted, where this toxic bloom has re-occurred each summer since 1985. The revenue loss to date has been about $10 million.

In mid-October 1987, the anomalous toxic bloom of Ptychodiscus brevis, which has now become so famous in its implication with dolphin die-off, occurred off Cape Hatteras. Paralytic shellfish poisoning occurred and 50 percent of the scallop population, I am told, and an estimated $25 million revenue loss was incurred by the fishing and tourist industries.

Clearly, these representative examples indicate that the coastal waters of the United States are likewise exhibiting an increased incidence of nuisance phytoplankton blooms carrying serious revenue loss and health hazard problems.

There is presently considerable scientific alarm, confusion, and uncertainty regarding the nature, causes, and regulation of the global epidemic and spreading of nuisance phytoplankton blooms.

This reflects the historical scientific approach to treat such blooms as rogue blooms, restricting their investigation superficial anecdotal descriptions of the occurrence.

It is interesting. In testimony this morning, one of the answers was that, well, we will study the impact. Very rarely do we get to approaches where we try to evaluate the triggering or causative factors in such kinds of things.

We tend to be impressed with the more sensational aspects of red tide blooms and other blooms, spectacular marine animal die-offs such as the dolphin, human illness and death resulting from paralytic shellfish poisoning, anoxic outbreaks, and development of odorous hydrogen sulfide, remarkable water discoloration displays, bioluminescence.

Finally, our reliance on the anecdotal "rogue bloom" approach has led to our inability to explain the causes of the global nuisance bloom outbreaks; to predict outbreak locations and periods; to account for the spreading phenomenon; to explain the sudden transformation of benign species into toxic ones; to account for local outbreaks.

It has also led to our failure to formulate sorely needed testable hypotheses upon which to design much-needed field and experimental research into nuisance blooms.

This has tended to perpetuate the anecdotal approach to such blooms and leads us to the results where we are this afternoon.

Equally important, this situation has precluded scientifically sound debate and inquiry as to the extent to which the global epidemic of such blooms is primarily an anthropogenic event, or triggered by natural, long-term variability, and trends in climatic and hydrographic patterns. If primarily anthropogenic, for example, what factors are specifically responsible generally, and for a given region?

A striking aspect, to me, of the nuisance bloom epidemic is its co-occurrence with the well-documented planetary trends in and stresses of acid rain; the greenhouse effect; increased ultraviolet irradiation accompanying ozone layer destruction; deforestation; changes in riverine nutrient loading and delivery to coastal environments; and coastal eutrophication.

Each one of these global patterns causes an environmental change that can in fact promote the growth of the phytoplankton. We ask, is there a linkage between nuisance blooms and these other planetary trends and stresses?

We cannot even begin to address this first-order question until we have a better understanding of nuisance bloom phenomena.

It seems clear, to me, that we have an ongoing equivalent of a "silent spring" in the sea, and that such parallel catastrophes as the dolphin die-off are a further manifestation of this aberration and must be viewed, must be viewed in this context.

Consider the fact that the phytoplankton have occurred in the sea for more than 3 billion years, where they have evolved, adapted, regulated biogeochemical cycles, and have served at the base of the foodweb.

The resilience of this remarkable group—in other words, their ability to bounce back from environmental assault or stress is very well known. They quickly go back to a normal pattern.

However, is the increased global frequency of their anomalous bloom dynamics and the greater emergent significance of nuisance species, and associated ecosystem dysfunction an indication of their loss of resiliency?

That is, should we consider such events—the Ptychodiscus bloom and everything else that follows from it—as the ultimate "miner's canary"? That the dysfunctioning of this ancient but major biotic component of Planet Earth is a particularly notable symptom, that our planet and its oceans are being pushed to its ecological limits prior to even more serious dysfunction?

Should we ask that question? I am hopeful that the Committee on Merchant Marine and Fisheries will find this information useful, within their purview and interest, and that it will evaluate this matter further, and then submit appropriate legislation to better understand and to remedy such deterioration of our ocean, its biota, and its ecosystem. Thank you.

[The statement of Mr. Smayda may be found at end of hearing.]

Mr. FOGLIETTA. I thank the gentleman. What I would like to do is to have all of the scientists testify and then we can ask questions, with one exception. The Ranking Minority Member must leave in a short while for another very important meeting, so I will allow her to ask the questions that she wants of Dr. Smayda, before leaving.

Miss SCHNEIDER. Thank you very much, Mr. Chairman, and the indulgence of my colleagues. Dr. Smayda, you seem to be saying in your testimony, or I should—I am so anxious to hurry up. Let me say, welcome, first of all.

Dr. SMAYDA. Thank you.

Miss SCHNEIDER. I am honored to have you testify before our Committee today.

Dr. SMAYDA. Thank you.

Miss SCHNEIDER. But you do seem to be saying, in your testimony, that we are already seeing the effects of global climate change, and other global dysfunctions in the ocean environment. Is that correct?

Dr. SMAYDA. That is correct.

Miss SCHNEIDER. And you mentioned that perhaps we should consider the proliferation of algae blooms as some type of ultimate "miner's canary". But in light of what we are hearing today perhaps we should be considering the Atlantic bottlenose dolphin as the ultimate "miner's canary". Would that be your ultimate conclusion, too?

Dr. SMAYDA. No. I think that the ultimate "miner's canary" will probably be regionally specific, in that while it may be the bottlenose dolphin in this particular area, it may be the salmonid and herring fisheries, say, elsewhere.

In a way, it is the "miner's canary," Congresswoman, except that rather than being a universal "miner's canary," one has to look at it on a case by case——

Miss SCHNEIDER. A regional way. I see.

Dr. SMAYDA. A regional basis.

Miss SCHNEIDER. Very fine. Well, that is the only key question I wanted to ask you, and I thank you very much, Mr. Chairman. Thank you.

Mr. FOGLIETTA. I thank the gentlelady. Now we will call on Dr. Beland, please. I would like to remind you: in the interest of time, I would ask if you could be as brief as possible. Try to hold your statement to five minutes, if possible.

STATEMENT OF PIERRE BELAND, PH.D., SCIENCE DIRECTOR, ST. LAWRENCE NATIONAL INSTITUTE OF ECOTOXICOLOGY, RIMOUSKI, QUEBEC, CANADA

Dr. BELAND. Thank you, Mr. Chairman, and Members of the Subcommittee. I really appreciate this opportunity to speak to an issue which has been truly remarkable.

My name is Pierre Beland. I am a research scientist, heading a team that has been investigating marine mammal deaths in the St. Lawrence estuary and gulf in Quebec, Canada, and I have been doing this for 6 years, now, and I really appreciate the amount of work that is involved when you have to deal with as many deaths as occurred on the Atlantic coast here.

And my review of the report is not addressed at the people who did the investigation, who, I believe, had a lot of work to do in a short period of time; but, rather, I am addressing the way that the data were selected for presentation, and the way that they were discussed.

And basically, my review of the report, I can summarize it by addressing four issues. The first one is, in my view, it has not been demonstrated that brevetoxin was the causative agent in this case, and I can name four examples, or four reasons why I believe that. There may be others.

One reason is that brevetoxins are difficult to quantitate, and this may influence the results, either way. I think there is a lack of reliable reference standards. The toxins are labile under extraction procedures, and there are losses in preparative steps, et cetera.

So it is not something that is easy to quantitate. If other labs attempted to quantitate that, they might come up with very different results for various reasons.

Secondly, there is no convincing evidence that the dolphins did indeed have access to sufficient amounts of contaminated fish. This evidence is not in the report.

Thirdly, there is no clinical data on the specific effects of acute or chronic exposure to brevetoxins. And the fourth reason is that there is nothing in the literature to suggest that brevetoxins caused some of the lesions, like chronic liver lesions found in the dolphins, nor that brevetoxin causes immunosuppression, and there was evidence of both in the dead dolphins.

The second point that I want to address is that the report is lacking, as far as evidence on lesions and on chemicals. For example, some types of lesions that would normally be found in such a large collection of dead marine mammals are not mentioned.

I am going to cite only one because it comes readily to mind, which is tumors. When you look at 300 animals, you should find at least one tumor. This is just an example, that some things have not been reported.

In the same vein, some organs were not reported from many animals, or some organs have not been reported on at all. And finally—and I think this is a very important point—thirdly, rather—no data are given on many chemicals of known toxicity.

The team has analyzed for PCBs and DDT, and chlordane-related compounds which, in the field of contaminants in our modern world, is what you would call "run-of-the-mill" things.

No effort was made to look for PAHs or dioxins, or furans, or dozens of other chemicals that are probably present in that environment, and very likely present in the dolphins as well.

Fourth, the results specifically on the organochlorine analyses are not very informative in view of what we know about such chemicals. In particular, the discussion specifically with regards to control animals does not fully consider age differences between animals.

It is well known that concentrations of PCBs and DDT are related to each other. These compounds travel together through the food chain, and they have the same solubility in lipids. And also they accumulate with age, so when you want a suitable control you need animals of various ages and sex in order to come up with a nice interpretation.

And finally, if one is interested in PCBs, it is now known that the toxicity of PCBs, which are a family of compounds comprised of more than 200 different—what we call congeners, or forms of the molecule—we now know that a few congeners, which I call coplanars, are responsible for most of the toxicity of a PCB mixture.

Now normally, one would look for those specifically rather than come up with the total amount of PCBs relative to a standard.

A third point that I want to address, and I started addressing that already, is the paucity of suitable control animals. When you carry out a study as a scientist, you want to have controls. Animals that have nothing to do with the event, so that you can compare what happened to your experimental animals, your stranded dolphins, and other dolphins taken from somewhere else.

Now I do realize how difficult it is to find suitable controls, specifically when you are dealing with marine mammals. But when you do not have suitable controls, I do not think you can draw conclusions as strongly as the report does.

The controls are only a few already captive dolphins, or recently captured dolphins, from the same environment as those that were found dead and stranded.

I do not think you can draw conclusions on the event based from animals that are very likely from the same population.

And my fourth point is that other scenarios should have been evaluated, and that is what I find being remarkable as a shortcoming in the report, is that although there is so little hard evidence implicating brevetoxin, while there are large amounts, high levels of chemicals of known toxicity— namely, organochlorine compounds, PCBs, DDT—and at the same time evidence of the effects of some of those chemicals, that the report fails to suggest any alternative scenario to the one involving brevetoxin.

I suggest that a team, or a collection of scientists in various fields could suggest, or have come up with various alternate scenarios that then should have been investigated by the team, or by a larger team.

So, I feel that the search for the initial cause, that has triggered the chain of events, is still open. Many elements may be involved. Brevetoxin may be part of it. The organochlorines in the animals are probably very likely a part of it, but several other agents should have been investigated.

And in conclusion, Mr. Chairman, I believe that we still do not know what happened along the Eastern seaboard in 1987. Thank you.

[The statement of Dr. Beland may be found at end of hearing.]

Mr. FOGLIETTA. I thank you, Doctor. Dr. Vargo.

STATEMENT OF GABRIEL A. VARGO, PH.D., ASSOCIATE PROFESSOR, DEPARTMENT OF MARINE SCIENCE, UNIVERSITY OF SOUTH FLORIDA AT ST. PETERSBURG, ST. PETERSBURG, FLORIDA

Dr. VARGO. Mr. Chairman, Members of the Committee and Subcommittee, thank you for this opportunity. I am a member of the

Department of Marine Science at the University of South Florida, and for the past 6 to 8 years have been intermittently investigating the initiation and persistence of red tide blooms off the mouth of Tampa Bay, which is in essence my own back yard.

My area of training is also phytoplankton physiology and ecology rather than toxicology, so I will confine my remarks to a series of short summary statements. They are very general in nature, but I hope that can be used to raise other questions about aspects of this report, and about studies of red tides in general.

I think, initially, Dr. Geraci and his associates should really be recognized for their foresight and their efforts in organizing and coordinating this study. Something like this is not an easy undertaking.

And they should be especially commended for the foresight in looking for biological toxins as part of their suite of analyses. There is little precedent, as I understand it, for this in marine mammal deaths.

The scenario proposed in the report offered by Dr. Geraci for the involvement of brevetoxin, and a chain of events that led to this mass mortality, is plausible and it is feasible. Unfortunately, the number of samples upon which it is based is far too few and leaves it open to question.

I think that as has been mentioned by many other people here today, that additional analyses of samples for Pbtx-2, and other toxins, or their degradation products, should be done to either enhance or negate his hypothesis. As I read the report, the samples were only standardized to one specific toxin produced by the dinoflagellate, Ptychodiscus brevis. This particular species produces five to six other toxins, three of which are of some degree of potency, and several of the others may perhaps be breakdown products.

Dr. Geraci can address this much better than I. But in the table indicating what analyses were done, if one looks at that, there were control animals that tested positive for three bioassays—as I think was pointed out earlier today—and there were several other animals from within the affected area that also tested positive. But they did not have peaks that co-migrated with the standards.

I think this may be the result of the presence of similar toxins to brevetoxin, or may possibly have been other types of brevetoxins. In any case, additional samples have to be analyzed.

The finding of, in Dr. Geraci's words, "unprecedented" high levels of DDE, PCBs and other organochlorines in the blubber and liver of this species of dolphin is—and again in his words, "a sad commentary on the state of the environment along the eastern U.S. shore."

These compounds were not accumulated by these dolphins overnight. Accumulation had to be chronic. We should really ask the question: Would this mass mortality have occurred if these compounds had not been present?

The presence of Ptychodiscus brevis along the east coast of Florida and into North Carolina has been established. The presence of toxin in menhaden—again very few samples—since the fish is a filter feeder capable of removing phytoplankton in this size range directly from the water column, has also been established by this report. There are no other analyses extant that indicate that menhaden bioconcentrate brevetoxin.

Red tide blooms of P. brevis have been considered, with a few exceptions, as a Gulf of Mexico phenomena. The North Carolina bloom, and the possible involvement of this organism, combined with terrestrially-derived pollutants in the deaths of top carnivores emphasizes the potential problems we have with the degree of water quality on the east coast of the U.S.

You cannot continue to think of our marine ecosystem as isolated regions. One cannot apply statutes in New Jersey and not expect them to be applied in Florida or any other State.

Coastal and oceanic waters of the U.S. are a continuum. I think Dr. Smayda emphasized that when he mentioned the brown tide bloom in three areas within a given region of the Northeast; the proliferation of red tide blooms worldwide; the massive blooms in the English Channel and along the Norwegian coast. Waters completely circulate throughout the world. Species, as long as they have the capability of withstanding the environmental conditions in a given area, might survive. Transport is inevitable.

I think events that occur in one region are going to affect every other region, and in this particular case, I think any future study should encompass the entire system from the Gulf of Mexico through the entire Gulf Stream system, for an East Coast study.

I have a considerable number of additional comments, but those particular ones summarize my feeling about this report. I might add just one additional comment that had been made by one of my colleagues after reading the report. That, again, the possibility of brevetoxin involvement was plausible, was feasible, but at this point is a speculation. Thank you.

Mr. FOGLIETTA. I thank you, Doctor. Dr. Goodwin.

STATEMENT OF MELVIN GOODWIN, PH.D., COORDINATOR OF INFORMATION AND EXTENSION SERVICES, SOUTH CAROLINA SEA GRANT CONSORTIUM, CHARLESTON, SOUTH CAROLINA

Dr. GOODWIN. Mr. Chairman, Members of the Subcommittee, I will be brief. I am from the South Carolina Sea Grant Consortium. In response to inquiries from private citizens, we undertook an examination of the body of records from the East Coast marine mammal stranding network related to strandings from Maine to Florida from 1978 through September of 1988.

We had three purposes in mind in doing this investigation. First, to assess the scale of the bottlenose dolphin mortality in relation to past strandings. Second, to determine whether similar increases in stranding levels had occurred among other species of cetaceans. And third, to determine whether the 1987-1988 die-off was a true anomaly or whether it was an explosive peak in a trend of increasing bottlenose dolphin mortalities within the past decade.

This analysis involved information from a total of 2,984 cetaceans representing a total of 33 species during that study period. Those data indicated that the number of bottlenose dolphin strandings during 1987 and 1988 was unprecedented during the period in which systematic records of such events have been kept.

While about 125 to 250 cetaceans typically strand on the U.S. East Coast per year, nearly 800 cetaceans stranded during 1987.

Although the greatest impact was on bottlenose dolphins, 1987 was also the peak stranding year for harbor porpoises, Atlantic white-sided dolphins, and humpback whales.

Stranding numbers exceeding previous yearly averages began appearing in July 1987 in coastal New Jersey, Maryland, and Virginia. By August, the highest numbers of bottlenose dolphin strandings ever recorded during a single month occurred in New Jersey, Delaware, Maryland, and Virginia.

Strandings in these States decreased in subsequent months, but anomalously high strandings of bottlenose dolphins occurred in North Carolina in October, South Carolina during November, and Georgia during December.

The progressive increase in strandings reached Florida in December of 1987, and attained a peak during January and February of 1988. Unusually high rates of strandings continued in Florida coastal waters through May of 1988.

With respect to the questions posed at the beginning of this investigation, these data established that the scale of Tursiops stranding mortality observed in 1987 and 1988 was several times greater than in previous years.

The data established that similar increases in strandings occurred among several other cetacean species, and the data suggest that the 1987 to 1988 event was highly unusual and is not consistent with any discernible trend in strandings during the past decade.

We have four problems with the explanation that has been advanced. These have been dealt with to some extent, and I will comment only briefly on those.

First, the pathology surrounding previous instances in which brevetoxin has been implicated in deaths of marine mammals is quite different from that observed during the 1987-1988 event. There has been an outbreak of red tide in Fort Myers, Florida reported from 1982, that was implicated in the deaths of 41 West Indiana manatees.

Post-mortem examinations had quite different results from those that have been reported for the dolphins. We would particularly like to call the Subcommittee's attention to the skin lesions that have been reported in many cases of the dolphin strandings.

These lesions, in early reports, were reported to resemble chemical burns and have not been dealt with in the final report. My colleagues have commented on the absence of blooms of Ptychodiscus brevis coincident with the dolphin stranding. We will not comment further on that.

We want to call attention, though, to other potential causes that did not appear to have been sufficiently examined. Of particular concern because of implications to other species, including humans, was the possible role of point-source pollution. Forty-one percent of the bottlenose dolphin strandings during the mass mortality event occurred along the coasts of New Jersey, Delaware, Maryland and Virginia, yet this area represents only 19 percent of the linear distance from northern New York to the Florida Keys.

Forty-one percent of strandings, and 19 percent of the coast. We have a concentration there. In addition to numerous point sources of industrial contamination in this area, there are a variety of ocean disposal sites as well. These need to be evaluated, in depth.

Finally, a few data have been made available to us that indicate relatively high levels of PCBs in the tissue analyses of three dolphins from the mass mortality event, that were studied at the National Marine Fisheries Service Charleston Laboratory in 1987. These animals are identified in appendix I of the report by Dr. Geraci as not having been subjected to chlorinated hydrocarbon analyses.

In conclusion, these circumstances, that is, the unprecedented extent of the mass mortality event, reservations concerning the explanation that has been advanced, the slight extent to which other potential causes have been examined, and apparent inconsistencies among official reports prompts us to urge that further inquiry be initiated with broad representation from the scientific and technical community to do two things.

First, to identify potential causes of the mass mortality event that should be considered, and second, to apply the diverse technical expertise available within the research, commercial, and governmental community to provide an in-depth evaluation of each of these causes.

In offering this testimony, we imply no criticism of those agencies and individuals who had undertaken the difficult task of explaining the 1987 to 1988 dolphin mass mortality. But we do suggest that the task is not yet complete. Thank you.

[The statement of Dr. Vargo may be found at end of hearing.]

Mr. FOGLIETTA. I thank you, Doctor. Dr. Martineau.

STATEMENT OF DANIEL MARTINEAU, D.V.M., M.Sc., DIPLOMAT OF THE AMERICAN COLLEGE OF VETERINARY PATHOLOGISTS, DEPARTMENT OF AVIAN AND AQUATIC ANIMAL MEDICINE, NEW YORK STATE COLLEGE OF VETERINARY MEDICINE, CORNELL UNIVERSITY, ITHACA, NEW YORK

Dr. MARTINEAU. Mr. Chairman, I am honored to be here today to testify before this Subcommittee. I am a veterinary pathologist and I worked on strandings of beluga whales on the shore of the St. Lawrence River, and that is why I am aware of the tremendous amount of work, the inevitable frustrations involved in the examination of 740 dolphins dying in a such small period of time, over such a long shoreline.

I have the following comments on the final report prepared by Dr. Geraci, and stating that the strandings were caused by a biological toxin. This claim is based on the detection of brevetoxin in eight of 17 carcasses, and in four fish, all of the same species.

One of the four fish was in the stomach of one dolphin.

This is little evidence to support that brevetoxin was the major cause of the stranding. On the other hand, the facts support that polychlorinated biphenyls, PCBs, alone, or with other factors, had an important role in the strandings.

The reason for this, are summarized as follows: Of all the lesions found in the nearly 240 carcasses that were partly necropsied, none can be actually related with brevetoxin toxicity since the lesions caused by this toxin in animals are unknown. In contrast, many lesions found in the carcasses have been described in laboratory and in domestic animals intoxicated with PCBs.

Additionally, high concentrations of PCBs were detected in all carcasses that were analyzed. Severe septicemia with a variety of opportunistic bacteria and lymphoid depletion were indicative of profound immunosuppression. PCBs are strong immunosuppressors while brevetoxin is not recognized as such. Yet, the relation of these lesions and immunosuppression to high levels of PCBs is ignored in the report.

Lesions and immunosuppresions caused by PCBs have been extensively documented in laboratory and domestic mammals with PCB levels comparable to those found in the dolphins.

In contrast, the lesions caused by brevetoxin, if brevetoxin causes any lesions at all, are not known, once more. The existing studies about brevetoxin effects are concerned basically with the effects on live animals, that is, on vital functions of their organs.

Bottlenose dolphins are mammals. As such, they have the same basic metabolic pathways as other mammals, and are exposed to the same toxic effects.

Lesions consistent with chronic PCB toxicity were found in stranded dolphins that were examined while high levels of the same compounds were found in all carcasses that were analyzed.

They would possibly have been found also in fish, if fish in this study had been analyzed for PCBs, or as Dr. Beland mentioned, for other organochlorine that were not investigated here. These considerations were not mentioned in the report as well.

Dolphins have been exposed for thousands of years to brevetoxin, and most likely they have developed metabolic pathways to degrade it. By contrast, exposure of bottlenose dolphins to PCBs is recent, since these compounds are man-made and were unknown in nature until the advent of organic synthetic chemistry less than 50 years ago.

Other animals in which PCB toxicity has been studied had not enough of that period of time to evolve efficient mechanisms for eliminating or de-toxifying these compounds. Most likely, dolphins being mammals, are the same.

In view of all this, it is impossible to disregard an important role played by PCBs in these strandings, considering the high levels found in the dolphins, and their consistent effects in other animals. On the other hand, it is also impossible to dismiss that another toxin, such as brevetoxin, had a role in the strandings.

The report simply does not contain enough data to support such a role. For instance, brevetoxin was found in four fish of the same species, the menhaden. Is it always normally present in this fish species as a background noise? Menhaden contained in suitable control dolphins were not analyzed for brevetoxin. If brevetoxin would have been present in menhaden from suitable control dolphins, we would have had a part of the answer to our question today.

Fish were not analyzed for PCBs in the report. Was another toxin directly responsible for the strandings, or were PCBs alone sufficient? It is impossible to say. This event should be seen like a warning. Official agencies, obviously, were taken by surprise by the magnitude of this event, and this should not happen again.

We have to admit that there is a problem out there. I think everybody here agrees. We can see this event like a single picture from a movie with a complex plot. In order to understand the plot we need more pictures.

A long-term monitoring program would provide these pictures. Such a program would include, for instance, a systematic autopsy of each stranded carcass with systematic sampling of liver, kidney, blubber, for organochlorines and biotoxins.

I would recommend to broaden the spectrum of the organochlorine compounds analyzed. The second part would be—and this is much more difficult I think—regular examination of captured animals to evaluate blubber thickness by non-invasive techniques. Such techniques, as ultrasounds, are available now for domestic animals.

Blood sampling of live animals to evaluate key functions of immune system, and to determine serum levels of organochlorines and biotoxins would be also important. I think with this we would have the elements to answer the questions we address today.

Blubber biopsy should be done on live animals for analysis for organochlorines and biotoxins, if the people present in this room want to have answers to the question we are addressing today. Only then could we talk confidently, and I hope correctly, about the roles of biotoxins in dolphin deaths, and probably of organochlorine as well.

If such a program was not implemented, a similar event would still take official agencies by surprise, and would lead to the same uncertainties that we are facing today. I thank you very much.

[The statement of Dr. Martineau can be found at end of hearing.]

Mr. FOGLIETTA. Dr. Smith, I will not say that we saved the best for last, but you do work in my district at Jefferson University Hospital. So you may proceed.

STATEMENT OF HARRY L. SMITH, JR., PH.D., PROFESSOR OF MICROBIOLOGY, HEAD, VIBRIO REFERENCE LABORATORY, JEFFERSON MEDICAL COLLEGE, THOMAS JEFFERSON UNIVERSITY, PHILADELPHIA, PENNSYLVANIA

Dr. SMITH. I was wondering if you were going to bring up that particular point. I thank you for the invitation to testify at this hearing.

Dr. Geraci and co-workers are to be congratulated on the report of the epidemic—actually, it is an epizootic—among the bottlenose dolphins. The logistics and coordination needed for this were tremendous and I salute the effort.

Collection of data is one thing; interpretation is another matter. Much depends on the background of the interpreter as to the conclusions reached. The methods and materials are not questioned, merely how one uses them.

To understand my interpretations, know that I am a teacher and a medical microbiologist working with a group of bacteria found in salt and brackish water, which causes diseases in a number of animals, including man.

The organisms, known as vibrios because they appear to vibrate when one looks at them in a microscope, are a part of the natural environment of dolphins and other marine mammals.

The report concludes that the brevetoxin from organisms that cause red tide "probably was the proximate cause of this devastating event". This is based on finding toxin in the livers of eight of 17 animals, or 47.1 percent. The bacteriologic data from table five in the report was accumulated into the following groups based on a general classification scheme. I put the vibrios in one group; enterics, which are organisms found in the intestinal tract and associated with the intestinal tract, in the second group; and in the third group are rounded bacteria that are called cocci.

I put this information in the table that is in my report. Vibrios constituted 168 of 322, or 52.2 percent of the isolates; enterics, 127, or 39.4 percent; and cocci, 27, or 8.4 percent.

What we do not know is the number of specimens taken nor how many were positive for one or more kinds of organisms.

Assuming an even distribution of the isolates among the 48 animals, vibrios could be implicated just as well as the brevetoxin as the cause of the disaster, based on percentages. The isolations could represent a part of the normal flora of either the animal or its environment, and does not necessarily mean that they caused disease in the dolphin. This is an area that needs research, as to the numbers and kinds of organisms in, on, and around marine mammals, and will be discussed later.

If we assume that the vibrios were responsible for the situation, how and why did this happen when it did?

As a part of the normal flora of salt and brackish water, vibrios must be able to survive and reproduce. Organic material must be present to provide a medium for growth. Under normal circumstances, the amount of organic material limits the numbers of microorganisms in the environment.

Animals tolerate normal flora as we are doing in the bacterial aerosol in this room. If the numbers of organisms increase, then the defenses of the individual may no longer tolerate the normal, which now becomes a pathogen. That is, it causes a disease. And I put in parentheses here: Immunity is relative—relative to the size of the inoculum that one is exposed to, and that applies to all animals.

With the dumping of sewage and other wastes into the sea, an enriched environment that promotes the growth of organisms such as vibrios could be created. The warmer the water, the more rapid the growth of the bacteria, perhaps explaining the geographic and seasonal incidences.

Now when the marine mammal swims through the area, it is like someone sneezing in your face. The inoculum is too great to be handled and the animal succumbs to the pathogen.

The mere presence of the organisms in increased numbers does not necessarily mean that the organisms can cause the disease. It must be something that makes it cause the disease. Vibrios have a number of factors, not well defined, that could be involved.

There is a toxin associated with the vibrios causing cholera in man that, at a molecular level, resembles the action of brevetoxin. We do not know the distribution of this or similar toxin in vibrios, or other organisms in the seawater environment. Vibrios cause severe ulcerative lesions when introduced into wound, such as those experienced by fishermen of both the fin and shell varieties. Even without lesions as a portal of entry, vibrios can cause disease.

My initial interest in the problem stemmed from a case in our hospital several years ago. A physician, on a snorkeling vacation in the Caribbean, developed a severe headache, was disoriented and could not walk straight. He returned home early to Philadelphia and was diagnosed as having sinusitis.

The material drained from the affected sinus was a pure culture of a vibrio. One assumes that the association with salt water was responsible for the introduction of the organisms. This was treated and he recovered without any ill effects. The disorientation of marine mammals in recent years brought this case to mind. If vibrios can cause a middle-ear infection in one mammal, why not in dolphins, with these animals losing their way and stranding themselves?

There is much conjecture in this scenario, which could be clarified through future studies. I would suggest the following: Study the microbial flora of salt and brackish waters, and the effects of environmental factors on the kinds and numbers of organisms. Measurements of physical and chemical properties can be done in conjunction with the biological studies. This should be done on a year-round basis to see if the numbers and kinds of organisms change. Include in this study the study of flora in, on, and around both sick and well marine mammals.

Two. investigate virulence factors of the organisms in the marine environment. Three, if sick dolphins or other marine mammals are encountered, pay particular attention to the organs of balance when collecting specimens for study.

My major concern is with the two-legged mammals that use coastal waters. I wonder if the dolphins are not like the canaries used in mines, to warn man of a danger that is present, but to which we are relatively insensitive. We may be doing ourselves and the dolphins a favor by learning about this problem before it reaches the point where people become sick and die. Thank you.

[A chart accompanying Dr. Smith's testimony can be found at end of hearing.]

Mr. FOGLIETTA. I thank you, Doctor. Now we will have questions presented by the Members, and the first questioner will be Congressman Pallone.

Mr. PALLONE. Thank you, Mr. Chairman. Dr. Vargo, I was interested in that section of your statement where you seem to say that a greater expenditure of energy during the migration of the dolphins could mobilize enhanced levels of PCBs from their blubber, making PCBs the primary agent for the dolphin mortality.

Could you explain that, and what kind of conditions would result in this greater expenditure of energy?

Dr. VARGO. I am not an animal physiologist. I am a botanist by trade. I was offering other possible scenarios that could have led to the mobilization of stored energy that is in their blubber—the fats, the lipids that are stored in the blubber—other than red tide, brevetoxin, or any other introduced toxicant. One possibility is if there were unusual current patterns involved. That is, as dolphins were required to swim either for longer periods of time, or at higher speeds than they would normally need to sustain.

Again, this is not my area of expertise, but that is one possibility. The problem here seems to be—or one of the problems here seems to be that there was a chain of events that occurred, not a single catastrophic introduction of a toxicant, but something debilitated them, initially. Then a secondary debilitation occurred as a result, or potentially as a result of the organochlorines, and the PCBs present in the blubber. As I understand Dr. Geraci's report, this is what he was presenting.

I was offering other explanations as to how the blubber may have been mobilized to release the organochlorines and the pesticides, et cetera.

Mr. PALLONE. In other words, the PCBs are in the blubber, and somehow, if the dolphins were using a lot more energy they would have to use more blubber?

Dr. VARGO. Right.

Mr. PALLONE. That is what we are talking about.

Dr. VARGO. In order to maintain migration, maintain their body temperature, it requires a certain amount of energy. If, for instance, they were not able to obtain food at their normal rate, well, they would have to burn up some of their fat, the same as we do, to keep going.

Mr. PALLONE. One of the concerns that I had—and I do not know if this question should be asked of you, or perhaps Dr. Geraci—is that there really was not much input from experts in the field of marine mammal behavior involved in the report. From what I can see, and specifically in our area, and Congressman's Hughes' district, we have the Marine Mammal Stranding Center with Bob Schoelkopf [phonetic] who was involved in trying to save some of the dolphins. I wondered why there was not input from people like him who are experts in the behavior of marine animals that might have given us a better indication of some of the hypotheses that you are putting forward.

Dr. VARGO. I would agree. I think there should have been, if there were not, but I am not capable of—I cannot really make that judgment, I do not know that literature nor do I know many of the people involved with those types of——

Mr. PALLONE. Thank you. Dr. Smith, as I understand it, you examined some of the early dolphins which washed ashore.

Dr. SMITH. No.

Mr. PALLONE. Oh, was it not you?

Dr. SMITH. No. Those were examined at the Atlantic City Hospital by one of my students.

Mr. PALLONE. So that you would not be able to give us any firsthand observation about those dolphins?

Dr. SMITH. Firsthand observations? No. I got the cultures and worked with them.

Mr. PALLONE. Thank you.

Mr. FOGLIETTA. Congressman Saxton.

Mr. SAXTON. I want to ask two questions. First, I want to ask you if the question that I am going to ask is fair.

Mr. FOGLIETTA. Excuse me. Would the gentleman yield. Would you address the question either to the panel, or to any particular doctor?

Mr. SAXTON. I am going to address it to the panel, if that is all right, Mr. Chairman. What I would like to ask you each to do—if you think this is fair—is to comment on one of these three statements, and to elaborate on the statement that you wish to comment on.

And I am not trying to pigeonhole anyone, but, as a layman, this is the best way for me to get a real sense of perhaps what you are saying.

First, the report is probably accurate and accounts for a logical explanation of dolphin deaths that occurred in 1987-1988; or, second, it is not reasonable to draw conclusions based on the data available from the study, and why; and third, it is reasonable to draw conclusions based on the data in the study other than those concluded in Dr. Geraci's study, and, if so, what would those conclusions be?

Is that a fair approach?

Dr. BELAND. Sorry, Congressman. I am still writing part of the first observation.

Mr. SAXTON. First, the report is probably accurate and accounts for a logical explanation of dolphin deaths.

Second, it is not reasonable to draw conclusions based on the data available in the study, and, if not, why not.

Mr. FOGLIETTA. Essay type? What do you want?

Mr. SAXTON. I just want each of them to respond to whichever one of these they would like to respond to.

Third, it is reasonable to draw conclusions based on the data in the study, other than those conclusions drawn by Dr. Geraci's study, and, if that is so, what are the conclusions that you would draw?

Mr. SAXTON. Mr. Hughes wants me to add number four: None of the above. Yes, sir?

Dr. GOODWIN. I feel like we are a little bit like the blind man trying to describe the elephant, because—I guess the most obvious example of what I mean is appendix I in Dr. Geraci's report, where it lists the individual sample numbers that were analyzed, and gives an indication of what analyses were done, but we do not have the results of the analyses. What we have are summaries.

I would find it very useful to be able to tell what the geographic and temporal distribution of some of the analyses were. We have reports that have been circulated in other parts of the literature, by other people involved in these studies, and, in some cases, quoting Dr. Geraci, saying, for example, that the animals, as time went on, were getting smaller or getting thinner, which might suggest—as Dr. Vargo suggested—that there was blubber mobilization.

And I would just like to suggest that we may be able to achieve some economy in terms of time and money simply by making all of those data currently available really available to anyone who wishes to look at them and comment on them. We may be able to get a much clearer picture as to what the possibilities are, from that exercise alone. Without having that, I do not feel we have enough information to answer any of the questions.

Mr. SAXTON. Dr. Smayda.

Dr. SMAYDA. I would selection option number three, that it is reasonable to draw conclusions based on the document data other than those drawn by Dr. Geraci, and the conclusion that I would come to is, one, brevetoxin was not the cause of the bottlenose dolphin die-off.

But the tabulated data presented partly by Dr. Smith, and other information here, suggests, if anything, that there was a general mosaic associated with, perhaps, exotic organic chemicals together with bacterial infection, which upon entering into the tissue systems of these different dolphins led to an epizootic which went through the community.

Some members of that community may have died because of vibrios; others might have died because of toxicity due to de-blubberization.

I believe all things point to a paradigm suggesting several concurrent events within a population that became stressed, for whatever reason, but that brevetoxin was not the major determinant of the die-off.

Mr. FOGLIETTA. Dr. Vargo.

Dr. VARGO. We started off by this panel, six scientists agreeing with one another, basically, which is almost unprecedented as well. I think we are getting away from that at this point in time. I think I would use some of Dr. Smith's arguments, and take your question number two, that it is not reasonable to draw conclusions based on the data available in this report for the point that was made by Dr. Goodwin, that we do not really have the data. We have summaries of the data.

I personally feel that there could be a complex mix of scenarios here, as Dr. Smayda does as well. Since there does not seem to be enough evidence to pinpoint a single prime cause for an initial debilitation, or an initial causative agent, then everything else was just a shotgun approach, and I think we are still at that point because dolphins, (a) are not continuing to die off, thank goodness, but red tides are continuing to happen.

So the involvement of brevetoxin, while possible, just really needs additional confirmation. I think that confirmation has to start in the Gulf of Mexico where we had red tides normally, and where we do not have dolphin die-offs, normally.

So I would choose reason number two, that it is not reasonable to draw the conclusions based on the data available, and we will see where we go from there.

Mr. FOGLIETTA. I thank you, Doctor. Dr. Martineau.

Dr. MARTINEAU. Yes. I agree with this previous opinion, that data is incomplete, and about brevetoxin, there is simply not enough data to support it as a primary etiology of this event. In case of PCBs, I think that they had a role, but their relative importance cannot be determined at the present time. And there is a possibility of a third toxin, a third organochlorine of which the presence is not reported, but of which the presence should be looked for.

So I am inclined for number two.

Mr. FOGLIETTA. I thank you, Dr. Martineau. Dr. Beland.

Dr. BELAND. Yes, Mr. Chairman. I would also select option two, but if I had to definitely come to a conclusion, I would opt for number three.

Mr. FOGLIETTA. Dr. Smith, please.

Dr. SMITH. Having taught medical students for 37 years, I would give the answer of "all of the above." First of all, the data, as presented, I believe are accurate, and I think Dr. Geraci is to be complimented on presenting the data, and as truthfully as he has.

Secondly, I do not think that it is reasonable to draw the conclusion that brevetoxin are the responsible agents for this.

Third, I think that you can draw a conclusion, such as I did, from the data that are there, but the whole point of it is that you take—when I say "all of the above", what I am saying is that what we have here is a good start for future studies on this.

Mr. FOGLIETTA. I thank you, Doctor.

Mr. SAXTON. May I follow up with one quick question?

Mr. FOGLIETTA. Wait until will come around again. Congressman Hughes.

Mr. HUGHES. Thank you very much, Mr. Chairman. Dr. Smith, perhaps I can ask you, and if anybody else wants to comment on it, they can. Was there a sufficient sampling?

Dr. SMITH. I have no idea.

Mr. HUGHES. You cannot make that determination?

Dr. SMITH. I could not make that determination.

Mr. HUGHES. Does anybody else want to differ on that?

Dr. MARTINEAU. I think there is a lack of sampling on suitable control animals, like fish found in normal, live, dolphins. If such fish would have contained brevetoxin, that would have answered the question. As well as their content in PCBs or other toxin, and what the dolphins ingested and toxins in the fish they ingested. That was not investigated. The fish were only investigated for brevetoxin.

Mr. HUGHES. Do I understand your testimony to be that in mammals and other marine life, found containing high amounts of brevetoxin, that lesions were generally not present of the sort found in the mammals, in the dolphins?

Dr. MARTINEAU. Animals dying of brevetoxin poisoning have never been examined in order to find lesions. The lesions are unknown. There is no study about the effects of brevetoxin on that aspect. That is simply the reason why I stated that.

Mr. HUGHES. We have talked about PCBs, DDT, and DDE. Can we rule out as a causative agent in the death of the dolphins other toxic organic chemicals?

Dr. MARTINEAU. No. You cannot rule it out because there is simply not information in the report about that. Maybe some other chemicals were there. There is evidence of toxic hepatopathy if you will read in the final report.

To my knowledge, the toxic responsible for that has not been found, simply not been found. The cause of this toxic hepatopathy is not determined as far as I know. It is an unanswered question.

Mr. FOGLIETTA. Can anyone, at this point, rule out the impact that dumping at the 106-mile site of all kinds of sludge containing heavy metals, PCBs, other contaminants——

Dr. MARTINEAU. As far as I know, a rupture of a container of a barrel, of a dozen of barrels are all possible, and these possibilities have not been investigated, or at least I see no trace of that in the report. If one suspects that a toxic compound is responsible for an epidemic like that, obviously one looks for the origin, for all possibilities of a toxic compound, and synthetic chemical compounds, are a possibility. I think there is not enough information.

Mr. FOGLIETTA. Do you find it curious that this phenomenon seems to have its origin about the time we began to dump at the 106-mile site? May of 1987.

Dr. MARTINEAU. I am not competent to answer that at all. I am not familiar with the dumping sites in U.S. I cannot answer that.

Mr. FOGLIETTA. Anyone? Any member of the panel?

Dr. SMAYDA. I think that the danger with such a kind of an exploration is that it gets it out of a regional context. I think that what we have to do is step back and view 1987 as truly an extraordinary year. We had, for example, the sudden appearance of the toxic dinoflagellate off of Cape Hatteras in mid-October. We had the dolphin die-off. We had the whales dying at Cape Cod.

And do you realize, in November and December of 1987, in Cardigan Bay, in Prince Edward Island, 156 people became ill from eating mussels. A toxin introduced in the mussels by a form of phytoplankter not previous known before, dumolic [phonetic] acid, now called amnesic shellfish poisoning. Twenty-two people were hospitalized. Ten were in intensive care. Three died. Several still are showing long-term effects.

What I am saying, Congressman, is that it looks like something incredible, unusual, anomalous, if you will, was happening all along the seaboard from Prince Edward Island all the way to the straits of Florida in 1987. It may just be that there was an aggravated accumulation of local epizootic events which, put together in migratory animals, given the flowing of the current system, resulted in a deterioration, if you will—I do not mean to be anthropomorphic—but a deterioration with the result that we had die-offs, we had these maladies that we had not had before, and so on.

But if we have a point source, such as the 106 dump site, and we have got dolphins that are maybe migrating 1500 kilometers, we have other kinds of problems to put in. You are asking a magnificent question. It is very difficult to sort of, perhaps, give you the quantification that you would like, one way, or the other.

Mr. FOGLIETTA. Well, we are policy-makers, and we are not scientists, but it seems to me it makes good common sense, when you did not have anything like this previous to 1987, and the only thing we had done differently is to begin dumping tens of millions of tons of sewage sludge at the 106-mile site, that would certainly indicate to us we ought to take a much better look at, you know, what is taking place at that site.

Dr. SMAYDA. I agree. What I have done is looked at the literature for other reasons, and there are magnificent examples from the Seto inland sea in Japan, where chemical and both domestic, but primarily industrial wastes, led to incredible environmental deterioration and die-offs amounting to 100s of millions of dollars of yellowtail and different fish kills. The Black Sea. The Baltic Sea. The Wadden Sea off the Dutch coast.

And in May and June of 1988, at a revenue loss of $185 million, between Sweden and Denmark, and Sweden-Norway, there was an incredible toxic bloom unknownst before.

In every instance, where you go into the data, where the data are appropriate, you do in fact see a build-up of nutrient in the form of nitrogen, in the form of phosphorus. There is no doubt that there is some kind of modification occurring with human activities in each of these areas that are in the direction of causing events of the kind we are talking today.

The nicest example, null hypothesis approach, is the Japanese industrial complex said, what would happen if we cut off our chemical delivery into the Seto inland sea, the amount of industrial wastes? The amount of red tides decreased, the number of fish die-offs decreased, and so on.

There is no question that, in a general sense, this modification is occurring, but to sort of get a "smoking gun", more or less, to say what it is in the 106 dump site—this is causing us all kinds of problems, and we scientists would love to have a congressional mandate with suitable funding, and suitable agency input, to help us to really come to grips before the event occurs.

All of our problems of the dolphin die-off, and all these, are events after the events have happened, so we do not know the triggering effects, and so we are backhoeing, best guessing, as to what is going on.

Mr. Hughes. Thank you. Thank you, Mr. Chairman.

Mr. Foglietta. Congressman Goss. Congressman Carper.

Mr. Carper. Thank you, Mr. Chairman, and to each of our witnesses, thank you very much for being here.

Dr. Beland, you indicated, I think at the end of your testimony, that we still do not know what caused the deaths of over 700 dolphins in 1987 and 1988. A question I would ask each of you to think about and respond to us, in writing, if you will, is: What further steps do we need to take, to determine with some certainty, what did cause the deaths of these dolphins, if you believe the conclusions reached in this preliminary study are inaccurate?

Again, if you would respond to us, in writing, once you have thought through that, that would be very helpful.

[The information received may be found at end of hearing.]

Mr. Carper. A couple of specific questions. Dr. Smayda, you mentioned I think that menhaden as the only vector, and I think by "vector"—what do you mean, something like a "carrier."

Dr. Smayda. Carrier.

Mr. Carper. For brevetoxin to dolphin populations along the mid-Atlantic coast. Could there be any other fish, or some other means, that you are aware of, that might enable the brevetoxin to reach the affected dolphins?

Dr. Smayda. Oh, sure. The reason for focusing on the menhaden was because of, in Dr. Geraci's report, and also there tend to be concurrent migrations of the bottlenose dolphin and menhaden. But most certainly other kinds of fishes that are feeding directly on the plankton, or on other fishes. The menhaden is interesting because it is one of the fishes that can feed directly on this particular dinoflagellate rather than having to feed on zooplankton, animal plankton, and then fishes. So it is almost like a direct transfer from the——

Mr. CARPER. All right. Thank you. A second question, really, for anyone on the panel. Do you have any idea how long this brevetoxin might remain in fish or in dolphins, and still cause adverse effect to the animals?

In other words, is there something like a lifetime during which brevetoxin might remain active in migrating fish or dolphins? Anyone know about that?

Dr. VARGO. I cannot answer it directly. You really should talk to a toxicologist, but people who have exhibited symptoms of ciguetoxicity [phonetic] produced by a suite of toxins called ciguetoxin, produces a disease called ciguetera which you get from eating the flesh of top carnivore-type fish—snapper, grouper, et cetera. People who have developed these symptoms often have them for months and years because the toxin is still present. It eventually does get degraded, however, over the course of months to years.

But there is no information available about the length of time the toxin would persist in the menhaden, or any other food source of the dolphin, or in the dolphin itself.

Mr. CARPER. Would anyone else on the panel care to respond to that question?

[No response.]

Mr. CARPER. Dr. Beland, are you suggesting that we are unable to—I think you talked about quantifying brevetoxin—but are you suggesting we are unable to detect brevetoxin in these animals, or that we simply cannot determine how much might be there? Could you clarify what you were saying, please.

Dr. BELAND. Yes. Congressman, what I said was referring to Dr. Evans' suggestion earlier, that other people might want to look at the same samples and come up with brevetoxin data—how much brevetoxin is in a given sample.

From speaking with colleagues of mine—I am not in that field of brevetoxin—but the response that I had was that if anyone in North America is qualified, it is Dr. Baden, who did these analyses, and this person, who is very knowledgeable on other types of toxins, has suggested that very few other people could come up with as valid a result as Dr. Baden did.

In other words, they are so difficult to quantify, that if you do not have the suitable standard, if you are not very careful, and if you do not have much experience with this specific analysis, the results may not be valid.

Mr. CARPER. Just one quick follow-up question, Dr. Beland. Is it possible that what has been identified as brevetoxin, or is believed to be brevetoxin, could somehow be something else? Or is that just not feasible?

Dr. BELAND. Well, no, I do not think I can answer that. It is always a possibility, but, you know, one has to rely on the given lab, and knowing that Dr. Baden is qualified, I would suggest that if he said he found brevetoxin, it probably is.

Mr. CARPER. Merci beaucoup.

Dr. BELAND. Je vous en prie.

Mr. FOGLIETTA. Congressman Saxton.

Mr. SAXTON. Mr. Chairman, I do not think I have a question. My question went to a statement that Congressman Carper actually made, and that was to just say that it appeared to me from my last question that the panel was fairly unanimous with some, perhaps variation, that there was not enough information in the report to draw conclusions, at least as far as the panel members were concerned. And I think that is the point that Congressman Carper made, in asking what we need to do to find out what happened to the dolphins.

So that was the thrust of my other question, unless somebody wants to comment on that. That was the conclusion that I drew from what you said, as Congressman Carper did. Unless you wish to comment on it, I have no further questions.

Mr. FOGLIETTA. I thank you. Congressman Pallone.

Mr. PALLONE. I want to say, with regard to Dr. Smayda, that your comments about the red tide were particularly appropriate. I felt from the beginning that the impression being given out, through the press conference, or through the initial accounts of this report, that even if red tide was a contributing cause, somehow that was a natural phenomenon not related to pollutants in the environment.

I think you have pointed out that that is not necessarily so; that the more common occurrence of red tide, particularly along the Atlantic seaboard, is related to the fact that there are pollutants, and that we are in fact introducing so many nutrients into the ocean environment, so that even if red tide were the primary cause, it is, in a sense, pollution-related. I think that is an important point.

I wanted to ask two things. There is some question about the samples that are being used here. Yet, it is my understanding there are samples in tissues from the dolphins that were stranded during this epidemic that have been saved.

If we were to continue the investigation, or re-open the investigation, as I have suggested, are these samples still useful for the type of research that would have to be done relative to PCBs or chemical pollutants? Could those samples be used? Would they be of any benefit at this point?

Dr. MARTINEAU. Yes, they are, since they are very persistent compounds. They are advantageous, so you can trace them back a long time after the death of the animal.

Mr. PALLONE. Well, that is important. The other thing I wanted to ask—a general question to anyone who would respond to it—what are the implications for humans from the high levels of chemical contaminants that have been found in these dolphins, and that we are apparently finding in other marine mammals?

I guess that is a difficult question, and it is general, but are there implications for humans, from what we are finding with regard to these chemical contaminants?

Dr. MARTINEAU. Dolphins are top predators. They eat fish and as far as I know, we are all in the same situation.

Mr. PALLONE. Anyone else?

Dr. MARTINEAU. So it is a direct implication I think.

Mr. PALLONE. Anyone else want to comment on that?

Dr. BELAND. Well, I remember some years ago, it was very frequent that you would read articles saying that eventually human beings on this planet would feed mostly from the sea, from fish being grown or captured in the costal zones. What the dolphins may be telling us is that if you try and do that you may not live very long.

Mr. PALLONE. Thank you. Anyone else?

Dr. SMAYDA. I think, Congressman, that what is of interest here is, ordinarily, these catastrophes in the sea have been restricted to fish, but through Dr. Geraci's own involvement, we know the humpback whale and the minke whale in Cape Cod, they died. The bottlenose dolphin died. The seals in the North Sea.

We are beginning to have a level of dysfunction in the community, very, very high up in the foodweb, and the full consequences of this are not known, as to the extent to which our coastal waters are generally deteriorating. But it certainly has—if not health-hazard implications to humankind—it certainly is saying something about the state of the environment that may impact on us negatively in other kinds of ways. I think there is something very, very significant going on here, that I do not think we have fully grasped yet, and I think you people are on the right track.

Mr. PALLONE. Well, I appreciate your comments, because as I said in the beginning, I really feel that dolphins are very close to man, and therefore, that there are direct implications. Thank you. Thank you, Mr. Chairman.

Mr. FOGLIETTA. I thank the members of the panel. I thank the Members of the Committee, and others, for being here. As you know, the hour is sort of getting late. I know that we want to hear from Dr. Geraci. We want to question him at length. So therefore, what I am going to suggest—and I have discussed it with the Members of the Committee—is that we recess this hearing until tomorrow at 11:30 a.m. in this room. I appreciate Dr. Geraci's indulgence. He was supposed to leave this evening, but he has agreed to stay over until tomorrow. We thank you for that.

[Whereupon, at 5:15 p.m., the Subcommittee was adjourned, to reconvene on Wednesday, May 10, 1989, at 11:30 a.m.]

MASS MORTALITY OF BOTTLENOSE DOLPHINS IN 1987-88

WEDNESDAY, MAY 10, 1989

HOUSE OF REPRESENTATIVES,
SUBCOMMITTEE ON OVERSIGHT AND INVESTIGATIONS,
COMMITTEE ON MERCHANT MARINE AND FISHERIES,
Washington, D.C.

The Subcommittee met, pursuant to call, at 11:40 a.m., in Room 1334, Longworth House Office Building, Hon. Thomas M. Foglietta (Chairman of the Subcommittee) presiding.

Members present: Representatives Foglietta, Pallone, Schneider, and Saxton.

Staff present: Phil Rotondi, Nancy Tyson, Chris Dollase, Mike Haas, Peter Marx, Brook Ball, and Kurt Oxley.

STATEMENT OF HON. THOMAS M. FOGLIETTA, A U.S. REPRESENTATIVE FROM PENNSYLVANIA, AND CHAIRMAN OF SUBCOMMITTEE ON OVERSIGHT AND INVESTIGATIONS

Mr. FOGLIETTA. Dr. Geraci, thank you for making the time to come back this morning. As you could see from yesterday's session, the Members of this Committee are very concerned about the environmental quality of our oceans and this opportunity to discuss with you your conclusions on the 1987 epidemic is greatly appreciated.

Before we begin, I do want to clarify the format of this hearing. Protocol would have had you testifying with or immediately following Dr. Evans. However, we did not want the science questions to become entangled with the legal and policy issues which we raised with Dr. Evans.

Also, because there have been different interpretations of your data within the scientific community, the Subcommittee wanted to afford you the opportunity to respond directly—after an airing of some of the differing scenarios and other questions.

With no further ado, then, we will begin. I understand you are accompanied by Doctors Ford Cross, Frank Ross, Karen Schlater, and Frank Pearson. Is that correct?

Mr. GERACI. Linda Schlater, yes.

Mr. FOGLIETTA. Linda?

Mr. GERACI. Yes, and James Pearson.

Mr. FOGLIETTA. I understand, also, that you have no prepared statement.

Mr. GERACI. That is correct.

Mr. FOGLIETTA. Before starting with questions, however, I would like you to begin by responding to some of the points raised yesterday and by any other questions that you know have been raised concerning this report of yours.

STATEMENT OF J.R. GERACI, V.M.D., Ph.D., PROFESSOR/WILDLIFE DISEASE SECTION, DEPARTMENT OF PATHOLOGY, ONTARIO VETERINARY COLLEGE, UNIVERSITY OF GUELPH, GUELPH, ONTARIO, CANADA; ACCOMPANIED BY P. FRANK ROSS, ANALYTICAL CHEMIST, NATIONAL VETERINARY SERVICES LABORATORY, ANIMAL AND PLANT HEALTH INSPECTION SERVICES, U.S. DEPARTMENT OF AGRICULTURE; DR. LINDA SCHLATER, HEAD, GENERAL BACTERIOLOGICAL SECTION, DIAGNOSTIC BACTERIOLOGY LABORATORY, ANIMAL AND PLANT HEALTH INSPECTION SERVICE, U.S. DEPARTMENT OF AGRICULTURE; AND DR. JAMES PEARSON, CHIEF, DIAGNOSTIC VIROLOGY LABORATORY, ANIMAL AND PLANT HEALTH INSPECTION SERVICE, U.S. DEPARTMENT OF AGRICULTURE

Mr. GERACI. Thank you, Mr. Chairman, Members of the Subcommittee. I would like to introduce, perhaps, more casually my colleagues seated with me.

Dr. Linda Schlater is head of the microbiology laboratory at the United States Department of Agriculture Veterinary Services Laboratory in Ames, Iowa.

Dr. James Pearson is the Chief of the Virology Laboratory at the Department of Agriculture Laboratory.

To my left is Frank Ross, who is the Analytical Chemist with the Department of Agriculture, and Dr. Ford Ross, Director of the Beaufort Laboratory, southeast region of the National Marine Fisheries Service.

I pronounce my name Geraci, Joseph Geraci, though, Congressman Foglietta, my grandmother would have noted with gracious approval your pronunciation of Geraci.

Mr. FOGLIETTA. I thank you.

Mr. GERACI. I am Professor in the Wildlife Disease Section, Department of Pathology at Ontario Veterinary College. I have worked 26 years professionally with marine mammals. My research and teaching focuses exclusively on dolphins, whales and seals.

I have led investigations into physiology, medicine, toxicology and stress-related studies for the National Marine Fisheries Service, Department of the Interior, Marine Mammal Commission, Office of Naval Research, Worldwide Life Fund and Canadian Natural Research Council, Canada Department of Fisheries and Oceans and the Canadian Department of the Environment.

Our studies focus predominantly on natural mortality, factors underlying natural mortality, stress and how these in turn relate to maintaining marine mammals in captivity.

I have no prepared testimony. What I would like to do instead is to present a very brief story and tell you how we proceeded with the investigation, the factors leading to the hypotheses and the supporting data for the various elements of the study that we undertook.

In August of 1987, I was asked by the Marine Mammal Commission to undertake the investigation of the cause of mortalities of bottlenose dolphins along with mid-central Atlantic Coast.

By that time, there had already been 30 or so dolphins that had died in New Jersey waters. We went to Virginia Beach because the heart of the operation was now centered there. The Smithsonian Institution was headquartered there, and they were collecting carcasses. Dr. Mead from the Smithsonian called me and asked if I would cooperate with their effort, and with the support of the Marine Mammal Commission I proceeded.

I arrived on Virginia Beach in August, August 7, and that afternoon I saw my first dolphin. We went to a small Naval base, and there a dolphin had been cast ashore. I had never seen a dolphin in that condition.

The skin was peeling. It appeared, as I wrote in my memo, as though the animal had been dipped in acid. The skin could be peeled as easily as a covering of cellophane.

When we examined the animal internally, some of the findings were very inconsistent, nothing I had ever seen before. This was not a typical condition; this was not a typical bacterial condition that I could recognize, nor a viral one; and we were very concerned.

We proceeded with those investigations, and through the course of that week, we had many more dolphins come ashore, more than we could comfortably handle. I knew we had a major problem on our hands, and I called the Marine Mammal Commission and various other Federal agencies to conscript all the support I could for this study.

By that point, we had a number of hypotheses, one that the animals might have been killed by contaminants; another that they had been facing a very hot, virulent agent of disease of some description. Those seemed to be the two probable causes of the mortality that had—that was occurring to the extent that this one had seemed to be progressing.

So we immediately went to the Department of Agriculture, and I went there because I felt comfortable with that laboratory. They look after the health of the Nation's livestock and the health of the, or safety of the food on the table; and I felt, perhaps, we should go right to that laboratory.

We were concerned about contaminant levels, so we asked that laboratory to undertake a broad range of studies on contaminants, given the limits of the studies that we had to do.

At the same time, we went to their virology laboratory and to other associated laboratories of the Eastern Virginia Medical School and since then to the National Institutes of Health, to conscript the aid of virologists to help uncover the cause of the problem.

We went to three agencies to work with bacteria. In fact we have the collaboration of three or four additional universities that have been working with us on the problem of identifying bacteria and trying to determine what role they might have had in this outbreak.

We were concerned and there was a lot of justifiable concern by people on the beaches. These animals were dying, and they were

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coming ashore in rather grotesque form. Folks were justifiably asking whether their children might be expected to come ashore that way after going swimming in the same waters.

We had reports of people who had problems because they were breathing the air or the shoreward breezes. So we knew that we had to address those concerns which we would have done in any case, but there was additional concern because we had early on said that we may be dealing with a problem that looks as though the animals may have had a problem with their immune system.

Immediately we, the world, thought of AIDS. Of course, we also tested for AIDS viruses in our entire protocol.

Well, we can summarize the results of some of these findings. We have generated a broad range of bacteria. We have discovered a broad range of organisms in these dolphins. The organisms are typically those that they live with. These are marine bacteria that you and I swim with when we swim in oceans throughout the world.

The dolphins are always exposed to them. But something triggered them to infect. Something made the dolphins susceptible to these bacteria that are normal inhabitants of their environment. So the finding of bacteria was just not enough. We could not conclude, because we know the nature of these organisms as well, that these alone were responsible for these dolphin mortalities, though indeed the dolphins eventually died of bacterial disease. That is clear. It is clear in the microscopic analysis of tissues.

We see the bacteria, we know where they are, we culture them from organs in the animal that we know are critical to their survival, and we can identify the hallmarks of bacterial disease. So we know many of them died associated with those organisms.

We also found viruses. We tested for a broad range of viruses but only found two or three, and we are continuing to characterize those viruses. There again, we know there are virus diseases in dolphins, very much like chickenpox, in fact, which affects them when they are stressed.

We saw ample evidence of that kind of disease in the dolphins. There again, it didn't seem as though a viral disease could be spread through the dolphin population and cause the devastation that we were seeing.

It is possible in seals. We were the team that found influenza, in fact, in seals in 1980 in Massachusetts. At least 500 seals died at that time very quickly after contracting an influenza virus a virus probably from birds. We know how that happened. Seals breathe on one another, they lie on one another, they sleep together on rocks, and it is easy to transmit a virus from one to another.

Not so with the dolphins, however. Dolphins don't rub shoulders. They have small family groups we know, but it is very difficult—it would be difficult for me to postulate a mechanism whereby a virus could as easily be transmitted in water as it can on land. So that was not among our more plausible causes of mortality.

The animals were not dying of a uniform disease, one that you could attribute across the board to a single infectious organism.

Then we went on to other factors, contaminants. We found in our selection of tissues from 80 animals that they have high levels of organochlorine contaminants, those of the DDT, DDE group, and we all heard yesterday of PCBs. In fact, they are among the highest of any of the animals on record.

I was sufficiently concerned about the quality of the data, that we then generated a number of control studies. We went to populations of animals that had absolutely nothing to do with these dolphins and went to the same laboratory and determined whether that laboratory universally comes up with high figures.

Well, they don't, because we had humpback whales, pilot whales, we had porpoises, great numbers of controls in order to test the veracity of the laboratory. The laboratory came out clean. As a matter of fact, we took samples and cross-labeled them so that the laboratory didn't know that they were in some cases analyzing the same tissues or tissues from the same animal, and I was satisfied and continue to be that we are going to have healthy livestock, and I am secure in eating at the dinner table.

In fact, without any reservation for the quality of the data, we are left with one conclusion, that these dolphins are carrying high contaminant burdens.

Now are we dealing with a condition which might have been precipitated by contaminants? If that were so, what would one expect to find?

Well, we need a trigger. Is it sufficient to carry contaminant loads in a dolphin or accumulate contaminants in the environment for 10, 20 or 30 years, and then all in one season die along with all of your mates in the same ocean on the same area? Not very likely.

Less likely when we know that the dolphins that have had high levels of contaminants died next to dolphins which had very low levels of contaminants. Contaminants alone cannot be postulated as the reason for these dolphins having come ashore, nor do we know precisely what these contaminants do in dolphins.

In fact, we know not at all because the literature on the effects of contaminants varies with the species, varies with the contaminant, and there is very little literature that would lead us in any direction except confusion if we try with any sense of credibility to associate the specific findings of these dolphins with contaminants.

As a matter of fact, we can, with some degree of comfort, associate the actual findings, the skin peeling, the pox lesions and so on, brain hemorrhages, with the presence of the bacteria that we isolated from these dolphins. So that is what eventually killed them.

Then we continued to look for a trigger. Something, somewhere had to start the process. During the study I went to Boston on a call that there were humpback whales dying in large numbers; and by the time I got there, in six days there were six whales dead. By the end of the month, there were 14 humpback whales and three minke whales that had died in Cape Cod Bay, a most unusual event and historically without precedent.

In those it was paralytic shellfish poisoning that affects people—never known to affect marine mammals—and we thought it a reasonable hypothesis that this might have contributed to the deaths of the whales.

We sampled stomach contents from the whales, found the toxin, and it took not very long in that case, three days, before we had three independent laboratories, including one from the National Marine Fisheries Service, confirm the validity of those findings.

Meanwhile, we looked for contaminant levels in the whales and found relatively low numbers, as we would expect in that species historically.

Armed with that information, we could not discount an earlier notion that we had that this entire event might have been precipitated by or triggered by a natural contaminant, one of the toxins which we know exist in those waters.

So I went to Dr. Steidinger, Department of Natural Resources. The organism responsible for the red tide was characterized, named by her. She is an ecologist who is thoroughly familiar with the red tide.

I went to her and sought her advice on the plausibility that this might be associated with a red tide event. She said, we don't know; we know there are a few red tides that have occurred in the East, a few, but nothing of any importance. I don't know where we could go with this. But she said, I will give you the name of the best person in the business who knows about brevetoxin, a toxin produced by the organisms, and that was Dr. Daniel Baden of the University of Miami.

I called Dr. Baden, and he said, I would like to get you off my back because I don't think that these toxins can be involved, but we will give it a try. I sent him samples. He didn't know which I had sent; they were coded, and only we knew where they had come from. We sent samples from the animals that died at Virginia Beach associated with the event, also animals that died in Florida also associated with the event months later.

We also sent control samples. We needed to verify whether or not if the animals have this toxin they just carry it for a living. For controls, we had material from captive animals, dolphins in captivity or that died in captivity, and dolphins that stranded on the coast of Texas and also dolphins that stranded precisely where these animals had but a year later, not associated with the same event but the same location.

The results of those studies, we sent 34 samples; I have been hearing 17. We actually analyzed 34, 17 control, 17 experimental. That was the limit of our ability to analyze samples within this time frame.

It is a very long and complex procedure, and from the time we started to the time I wrote that report, we had 17 experimental, 34 altogether.

Of the 17 animals which were controls, we found no brevetoxin. Of the 17 animals, an equal number, which came ashore associated with the epidemic or epizootic event, 47 percent of those animals had brevetoxin.

Now, what we do in these cases is weigh the evidence. We cannot conclude from a natural event why animals might have died retrospectively; that is impossible. It will never stand up in a court of law, and I wouldn't want to be challenged by any of you to defend such a statement. I could not.

What we do is weigh hypotheses. In science, the way we operate in that kind of event, in a natural event, is to get as much data as we can and then weigh the data for its evidence.

The evidence now would lead us to the most plausible cause of the trigger—not the reason why the animals died—but the trigger as being brevetoxin. The toxin was found in livers at concentrations which make people ill. We found it not only in the dolphins, but we found it in fish the dolphins were eating.

The events are entirely new. Dr. Baden was very surprised by the findings in both cases, and now we are together. Dr. Baden, Dr. Steidinger, and I just met on Monday, in fact, in a workshop on the effect of biological toxins on marine mammals, the first of its kind, held at Woods Hole at Cape Cod.

Those of us, 11 of us got together for the first time to see what is this all about. Whales are dying, associated with saxitoxin and clearly some of the dolphins have brevetoxin in levels harmful to other species. What effects does brevetoxin cause in a dolphin? I have not the slightest idea.

What effects does long-term accumulation of the same toxin cause in people that we know are poisoned when they eat clams contaminated with them? We have no idea, because people do not accumulate brevetoxin over long periods of time. It's without precedent.

It's also without precedent that fish accumulate the toxin. We know that the fish die in the west coast of Florida at times of red tide blooms, when the organisms are flourishing. Blooms are perceptible as a red discoloration of the water. The organisms produce the toxin, and sometimes fish die of the effects.

Well, now we know that fish don't always die of the effects because in fish that the dolphins were eating, we found levels of the toxin in the liver. So fish we know can be carriers, and we know that the dolphins are eating fish and subsequently that this material is transferred into dolphins.

The weight of evidence, again, is that we are postulating that the trigger that led to this event was the dolphins eating fish and accumulating brevetoxin—not that the toxin killed them. What we suggest instead is that the animals only became ill as people become ill, and not die. And when people eat this—eat clams and have in their bodies less total concentrations than the dolphins do, they have stomach aches, dizziness, heart palpitations, they have sensations of hot and cold reversed so you don't know if you feel something warm or cold. Ultimately they have respiratory problems, difficulty breathing and in experimental animals, they die of the respiratory effects. They become effectively paralyzed. This is a neurologic toxin.

What we are saying is let's not assume the animals died this way, but let's get a good belly ache into a dolphin. I know what a dolphin looks like with a belly ache because I deal with them in captivity. They don't like to eat, and go off their food. When they go off their food, they need to depend on blubber. Blubber is not only a source of energy as fat, but to a dolphin, it's a source of fresh water, very much like a camel depends on its fat in times of drought for its water; dolphins do as well.

When they utilize that blubber to obtain energy and water, the blubber gets thinner, and that is their source of buoyancy. Now, the animals need to struggle to stay at the surface much like a thinner person would have to struggle to stay at the surface of the water.

And that blubber is their thermal blanket. That is what keeps them warm. Now, the animal has lost the ability to stay warm, float effectively, and to have enough energy left to sustain itself when it is not eating. That is all we need for stress.

Now, if the animal is stressed, we can invoke any number of mechanisms whereby the animal will die. It matters not what bacteria and viruses are there to kill it, or what other metabolic diseases are there to do the same.

What of contaminants? We know contaminants are in the blubber of the dolphin. It's an inactive depo. To correlate contaminants with any more than a depo at the levels we see would be going beyond the credibility of the data that we have gathered.

However, when the dolphins start to utilize their blubber, we know and we show in our report that they mobilize the compounds from the blubber into the liver. Now, we have the contaminant in an active critical organ, no longer a storage depot but an organ that the animal needs to sustain itself, especially when it is not eating.

We know that the levels of—I am uncomfortable with the levels of some of these organochlorines we saw in the dolphins. We also know we only tested for a representative range of compounds. There are many more out there that we could spend lifetimes examining. That wasn't the issue. That wasn't the point.

The point was to see what actually happens with representative compounds. We know that they go into the liver, and we say without dismissing the importance of these contaminants, in our report on Page 16, "considering the evidence that at least some of the dolphins were mobilizing PCBs from blubber to liver, it is conceivable that blood levels rose and were sustained long enough to exert an effect."

We have gone beyond that to suggest what the effect might be. One class of organochlorines, the PCBs, can be harmful following both acute and chronic exposure. Typically affected are the liver and skin and nervous, reproductive and immune systems. Yet we cannot categorically relate any of the conditions observed in the dolphins to the known effects of these compounds because of vast differences in response between and within species.

We recognize the effects of these compounds, but again, it would be going beyond the credibility of our data to suggest that we have anything more than a correlation without being able to attribute or able to suggest that we have a cause. So this is not a cause and effect but a correlation.

The plausible scenario then is, to my mind, that the dolphins had—that this condition was triggered by a completely new occurrence of a red tide phenomenon and their feeding on fish that were contaminated with brevetoxin.

At that point, the animals became ill, and as they did, a number of things happened to them. They died of bacterial and viral diseases, and also we must add to the equation the possibility that contaminants might have weakened their organ systems and in some way made them more susceptible. We cannot deny the possibility.

I would like to conclude with a brief statement, if I may. I spend my life on the ocean. My professional career, I guess I call myself an ocean person. In some ways, I see the ocean as my front yard and my laboratory, and I also see dolphins a little different than perhaps we do traditionally.

Dolphins are wild species, wildlife. Yet we are so involved in their environment, we utilize the fish that they eat, we put contaminants on the dinner table, and we put an outhouse in their living room. We then have become their custodians.

Dolphins are really wards of the state. I think we have the same responsibility to them as we have to other species, in fact, captive species. As a matter of fact, the Department of Agriculture standards would not allow a dolphin to be living in the near shore waters of the Atlantic because in fact the regulations are so rigid that they would not allow a dolphin to be maintained in the pool that has rust exposed. So we know there are incongruities here.

I feel as we all do, that we have more contaminants in that ocean than we need. Certainly it's not good for the dolphins, and I would like to see it out of there. But I cannot use the data from this report, the data do not—I cannot go beyond the credibility of the data. I cannot go beyond the point at which I can make nothing more than a correlation between contaminants and the mortalities we are dealing with.

However tempting the urge, the data do not bear out that kind of conclusion. So we remain with the plausible cause as the triggering by the brevetoxin and mortality ultimately by infectious agents and somewhere in that scheme, contaminants may play a role.

Thank you.

Mr. FOGLIETTA. Doctor, I want to thank you for an excellent dissertation. It was detailed, it was thorough, and most important, it was easily understandable to non-scientists or laymen, lay people like ourselves.

I do have a few questions, however. Were any restrictions placed upon you in the conduct of your investigation whatsoever?

Mr. GERACI. None, sir, none at all.

Mr. FOGLIETTA. In a letter which you sent to Dr. Evans on May 10, 1988, there is a notation I would like you to explain. Below your signature you write in longhand, "Thanks again, Bill; we are on a roll now. Hope we will stop short of the cliff."

Would you like to look at that?

Mr. GERACI. No, no. I believe it. I do that quite often.

Mr. FOGLIETTA. I would like to know what the cliff is. We are puzzled as to what you were referring to there.

Mr. GERACI. I can tell you that at the time I wrote that letter, I was working 15- to 18-hour days, and I indeed was on a roll; and I did in fact stop short of the cliff or I probably wouldn't be here. In other words, I guess this was just a metaphor for I am going nuts.

Mr. FOGLIETTA. Well, thank you, Doctor.

How do you reconcile the migration of the dolphins and its prey species and the movement of the red tide bloom with your brevetoxin conclusion, particularly as an explanation for the deaths of the first 180 dolphins? From what we have heard yesterday and today, no bloom actually appeared in the Atlantic until three months after the first dolphin stranding.

Mr. GERACI. I would like to defer that question to Dr. Cross.

Mr. FOGLIETTA. Dr. Cross.

STATEMENT OF FORD CROSS, Ph.D., DIRECTOR, BEAUFORT LABORATORY-NATIONAL MARINE FISHERIES SERVICE, U.S. DEPARTMENT OF COMMERCE

Mr. CROSS. Thank you.

It is true, we don't have any conclusive evidence that Ptychodiscus, or the Florida red tide organism, was present in the Atlantic. A very large red tide came ashore on the North Carolina coast in late October 1987. However, we do have what we believe is very good circumstantial evidence that there was contamination or infestation of red tide along the east coast of Florida in the vicinity of the wintering grounds of dolphins and a number of migratory fish as early as the spring of 1987.

Starting in September, 1986, going backward chronologically, there was a red tide off the west coast of Florida, and it lingered for quite a while in through the winter. There were fish kills reported a number of miles offshore, and measurements were made of good concentrations of cells in the water as late as February 1987.

Anecdotal reports of fish kills offshore where the Gulf Stream—which is a Gulf loop current that becomes the Gulf Stream—transports cells around the Florida Keys along the east coast of Florida were coming in from fishermen.

So, there was a red tide off the coast of Florida in an area where there is active transport to the east coast of Florida.

With your permission, I would like to read a short paragraph that Dr. Vargo presented in his testimony yesterday considering the chance of there being infestation on the East Coast in this time. He said the arguments presented regarding low population levels of *P. brevis* going undetected in the water column have counterparts on the West Florida Shelf. Unless population levels are high enough to yield fish kills, they are seldom detected without a sampling program specifically designed to monitor for their presence.

It is my opinion that the red tide cells are transported from the Gulf of Mexico to the Florida current, which is essentially the Gulf Stream, whenever Gulf populations are present. Filaments of Gulf Stream that reach nearshore waters along the southeastern States also occur. It only remains for the proper physical conditions to develop in nearshore waters that concentrate cells and maintain populations in the discrete area long enough to produce a bloom.

P.Brevis does possess physiologic and biochemical attributes that allow it to persist and grow in the nutrient-poor waters of the Florida current and Gulf Stream.

We think there is good circumstantial evidence that there were cells transported to the east coast of Florida for an extended period of time during the winter of 1986-1987 to the wintering grounds of the dolphins.

We know six months later a massive red tide showed up on the North Carolina coast. What we do not know is the extent of east coast contamination in that time. We had originally thought that our red tide in North Carolina stemmed from an August-September 1987 red tide off the west coast of Florida, but it is entirely feasible that we had contamination low enough to contaminate the foodweb, but not high enough to cause fish kills, throughout the summer, which would expose the dolphins for an extended period of time in the southeast as they migrated northward.

Mr. FOGLIETTA. Your conclusion is it was of sufficient magnitude and intensity to have been the cause.

Mr. CROSS. It certainly could have been, yes.

Mr. FOGLIETTA. Dr. Geraci, at one point during the epidemic, EPA promised it would make its research ship, The Anderson, available. Then it was rerouted, and it was not made available. Was it ever made available to you, and is there data that you would like to have had from that ship?

Mr. GERACI. Yes, sir, it was made available to us; and we utilized that for a survey. At the end of August, we sent two members of our team with the ship, and they did an offshore survey. Ultimately we accompanied them on the survey to determine whether there were dead carcasses at sea. We were quite satisfied that most of the dolphins, many of the dolphins were coming ashore, too many, but we wanted to know how much mortality we were not observing.

So we went to sea where we could detect carcasses. We had a number of reports that came from aerial spotters and ships that there may have been dolphin carcasses. EPA was kind enough to provide the vessel, and we found no carcasses.

Mr. FOGLIETTA. Dr. Geraci, I have no further questions of you. Again, thank you for being here and giving us your thorough explanation. I want to thank the other members of the panel, also.

Congressman Saxton, please.

Mr. SAXTON. Thank you, Mr. Chairman.

Dr. Geraci, I want to first express my appreciation, and I am sure the appreciation of the other Members of the Committee, for your staying an extra day so we could spend this time together to help us better understand what it is that you have found and how you arrived at your conclusions.

It is very important for us to be able to address these problems and these situations as knowledgeably as we can, so we do thank you very much for staying this extra day with us.

When we passed the law which created the study, we indicated that we were interested in finding the extent to which pollution may have contributed to this epidemic. My understanding at this point is that you feel that the proximate cause of the dolphin deaths was brevetoxin; is that correct?

Mr. GERACI. Yes, sir.

Mr. SAXTON. To what extent do you believe that toxins may have played a role?

Mr. GERACI. You mean contaminants or——

Mr. SAXTON. Contaminants that were found in the blubber of the dolphins and later in their livers as well, from what I understand.

Mr. GERACI. Sure. Well, we see them there in high concentrations. We know that they are mobilized from the blubber to sites where they could pose a potential threat to the animal when the animals are losing—utilizing that blubber for energy.

So, I guess I am concerned that there may be some effect, but I have no—I can provide no information on what that effect could be in the dolphin nor do we know the extent, if any, that those contaminants might have played.

I can add to that, it is part of our control study that we took samples of liver and blubber from dolphins that had died in captivity, had been in captivity for years, and the levels of pesticides and contaminants are the same in those dolphins as they were in the dolphins that were dying at Virginia Beach.

So on that basis——

Mr. SAXTON. I have a general understanding, however, that in the case of the dolphins that you studied, that the levels of contaminants were the highest levels ever measured in dolphins.

Mr. FOGLIETTA. Sir, they were among the highest. I think we have one value which Congresswoman Schneider mentioned, 6,800 parts per million. That is really high. So they are among the highest, clearly.

Mr. SAXTON. Doctor, did you give any consideration to the fact that the 106-mile dump site was established and began to operate, I believe it was in March, the 17th of March of 1987?

Mr. GERACI. Yes.

Mr. SAXTON. And that—obviously that is a new source of toxins in the ocean which became a site for those toxins shortly before we began to see dolphins wash up on the beaches in all the conditions that we have described here over the last two days.

Do you find that a concern in any way, and if so, did you explore the possibility that that may have a related cause, and if so, how?

Mr. GERACI. Yes, I did explore that. We were aware of that. We requested from the EPA information on the 106-mile dump site. We received quite a bit of information on it. We got information, including a report, a paper in the open literature on the effects, projected effects that dumping in that site would have—that is trajectory patterns, dispersion of the contaminants, dilution factors and so on.

So we, as part of the exploratory effort, did examine the information on the 106-mile site, and on the 12-mile dump site, the old 12-mile dump site, as well.

Mr. SAXTON. I don't recall seeing that in your report. Was it in there?

Mr. GERACI. In the interim report we mentioned, yes, that we had asked for information on the 106-mile dump site. It is part of the—it is in the interim report.

Mr. SAXTON. So you are saying that you could find no correlation or no reason to believe that activity at the 106-mile dump site may have been a contributing factor to the dolphin deaths?

Mr. GERACI. I cannot say that, sir, but what I can say is we do look for evidence that it might have been. To do that, we know the character, the general blend of things, if you like, that are discarded, and so we examined the dolphins for evidence that they might have been involved or that they might have picked something up from that site or any other site. We didn't discriminate that site from any other possible point source of contamination.

Had we found something in that site that we were very concerned about in the dolphins, we would have explored it further, but the evidence was not pointing to any further investigation of that particular site.

Mr. SAXTON. Are you still convinced today that brevetoxin was the, I think as you put it in your report, proximate cause of the dolphin deaths?

Mr. GERACI. Yes, I believe with all the evidence we have, I believe it is the most plausible trigger for this event, again recognizing that there are a series of events that preceded the actual deaths of the dolphins.

Mr. SAXTON. Can you explain in an academic way the reaction that we got from the people that I would refer to as your colleagues on the panel yesterday who seemed to disagree with you?

Mr. GERACI. Well, most—mostly I was rather—I listened to I think it was Professor Vargo's statement that he had never been in a meeting in which there had been so much unanimity in a panel, and I guess I have not, either.

Mr. SAXTON. Excuse me. It seemed they were unanimous in saying they could not draw the same conclusion you did, based on the information.

Mr. GERACI. Yes, indeed. I wasn't postulating anything else. That is fine. But I think you may not have known the system by which we operated, so I would like to bring to your attention the system under which we operated with the peer review process.

I submitted the report, and it was the final report. I knew that it was going to be subjected to peer review. Under normal circumstances, one expects that peer review to operate using two or three external referees. As an editor of a journal, Marine Mammal Science, I sought two reviews. If I had a problem, I would seek a third.

I thought that we would go to two or three reviewers. The reason why there was such delay between the final report that some of you referred to and the ultimate final report that we have before us, which is a month later, was that that paper was refereed by 44, at least 44, and I think it was perhaps closer to 60 colleagues. So there were a number of scientific colleagues that reviewed that paper.

As in the way we normally operate, we take all of those reviews, we compile comments to find those that seem to be consistent among all reviewers, and we address every one of them. We addressed them in one of two ways; modify or revise the paper by admitting that, my gosh, I had missed that point, and it's a good thing it was brought to my attention, and I will address it now in the revised report; or, to rebut it in some way and say no, I think you missed the point, I should have made the sentence clearer but in fact this is what I meant.

So, that report that you read has been reviewed by 44, if not more, of my colleagues. The six that you saw did not appreciate the report, I don't feel good that forty-four others reviewed it, but I accommodated their comments.

I might add as well that if there was a general tone, it was that in my preliminary report—and I really admit that I had been too conclusive, that I had in fact established a plausible hypothesis, but I had been too conclusive in my comments. So I toned it down. Not toned down in the sense of changing my mind but making it a plausible hypothesis which is, in fact, what it was.

I don't recall any instance in which any of the reviewers suggested that I had overlooked contaminants or that contaminants played a greater role than we had in fact ascribed to the mortality.

Mr. SAXTON. If I am not mistaken, I think you have indicated that at least 44 people were part of the peer review group or there may have been more.

Mr. GERACI. Yes, sir.

Mr. SAXTON. Two of the people, I am told, on the panel yesterday were part of that group.

Mr. GERACI. That may be. I don't know. I am not supposed to know that.

Mr. SAXTON. It would be some indication that would indicate to me that perhaps the peer group wasn't unanimous at least.

Mr. GERACI. If you can get 44 scientists to be unanimous, I want to be at that party.

Mr. SAXTON. Mr. Chairman, out of curiosity, how does the peer group report to you? Not being a scientist or ever having written a paper similar to this, I wouldn't have any idea how they work.

Do they send you a record on their agreement, disagreement, or comments or dissenting views?

Mr. GERACI. In this case, yes, it was done—what was done was it, the paper, was sent to the National Marine Fisheries Service, and they in turn sought, identified the reviewers within and outside the Service, and the comments were returned to the National Marine Fisheries Service, and they were transformed so that they were anonymous. I did not know the identity of any reviewers, which is part of the process.

Mr. SAXTON. So, some disagreed; did some of the peer reviewers disagree with your conclusions?

Mr. GERACI. Oh, indeed, that has always happened. All my life I have had colleagues disagree with some things. That is part of the process.

Mr. SAXTON. I would just like to know if you could respond to one more question. Do you have any problem with releasing the data and the information that you may have used to draw your conclusions to other members of the scientific community at this point?

Mr. GERACI. Indeed not, sir. That is part of my contractual obligation with the National Marine Fisheries Service. All of our data is on disk, and that is submitted to the National Marine Fisheries Service as part of our contract. Also, I have made available—we are making available, now that the study is complete, making available all supporting data. It is a part of our contractual agreement to do so.

Mr. SAXTON. Apparently, Doctor, that information has not been available up to this point; however?

Mr. GERACI. Yes, sir, that is correct. I would like to draw your attention—may I read a statement that I submitted to Congress as part of the testimony in September 3,1987?

Mr. SAXTON. Certainly.

Mr. GERACI. Because there has been—I would like to clarify a point.

I think there has been some misapprehension about the fact that we maintained a pretty tight hold on the materials from the study and on the results of the analyses as they were emerging.

Our study plan was very specific in design. We intended not to release premature data. Premature data causes problems, premature data leads to fusion reactions in our basements. Until we can confirm among our scientific peers that the data we have are valid, we maintain strict confidence.

I designated in my testimony to Congress—I would like to read one paragraph, if I may.

> "As the investigation in Virginia Beach was being organized, it was recognized that the number of individuals, organizations and laboratories not associated with the response team might offer to provide assistance or request specimen material for independent analysis.
>
> "Responding to such requests would place an additional burden on the response team and could interfere with its mission. We also recognized that objective evaluation of the results of the investigation likely would require comprehensive evaluation of the results of the full range of bacterial, viral, toxicological and environmental studies being initiated and that providing samples to other laboratories for independent analysis could lead to premature or false conclusions. Such conclusions could jeopardize the merits of the investigation, particularly if the mortality was somehow related to illegal dumping of toxic waste or other human activities that could be subject to legal action. For these reasons, it was agreed by the involved Federal agencies that offers of help or requests for tissue samples would be denied, one, unless there was reason to believe that an individual laboratory offering to provide help or requesting tissue samples could provide a service not already available to the response team; or two, unless the investigation was concluded and the results made public."

The investigation was concluded last month, the results made public two weeks ago, and today all the specimen material is available for independent analysis, as would be all the data that we generated.

Mr. FOGLIETTA. I thank the Congressman.

Thank you, Doctor.

Congressman Pallone.

Mr. PALLONE. Thank you, Mr. Chairman.

Doctor, I appreciate your comments today because I think I have a better understanding now of your position and the conclusions and how they came about.

I am still very disturbed. If I listened to what you said and what the other scientists said yesterday, the only consensus I really can see is the possibility that PCBs or chemical contaminants may have been a contributing factor and that more data or more study would be needed to make that determination.

Some of the scientists yesterday, I think Dr. Martineau was the one that I most easily remember, specifically ruled out brevetoxin as a possible cause. Then there was Dr. Vargo who basically brought up, when I asked about it, the same point about the blubber and the PCBs or other contaminants in the blubber; that somehow blubber is used through a triggering mechanism that could affect the breakdown of the immunity and make the dolphins susceptible to diseases or other factors.

But I am concerned because the very thing that everyone seems to agree on—which is that contaminants may have been a factor—is the very thing that we seem to be needing more data on, that we seem to need more investigation of. I think you, yourself, said that there really isn't that much information available on what high levels of these contaminants can do to dolphins.

So, my initial question is, and this is what I was most concerned about, can we or do we need to do more investigations, as you say in the last sentence of your report, to resolve the growing question of whether contaminants at levels found in the dolphins might have affected their resilience and rendered them more susceptible to the toxin or micro-organisms that eventually brought them to their demise?

Do you feel we need more investigation of the contaminants and their possible relationship to all this? And I guess the second question is, are the samples that are available, is the data you have, can it be reanalyzed to draw some further conclusions in that regard?

Mr. GERACI. The material is available for analysis. The data are available for reevaluation, yes.

I did not hear Dr. Martineau say that the contaminants caused the problem. I don't believe he would say that. I think he might have said they can.

What I am saying is, that they can, as well, that they are involved in the problem—they might have been involved in the problem. Let me clarify that.

But I don't know the extent to which they were involved, nor do I believe Dr. Martineau suggested the extent to which they might have been.

Mr. PALLONE. I didn't suggest that Dr. Martineau had said that the contaminants were definitely the cause. He, as you, indicated that that was a possible cause and that more study needed to be done in that regard.

What he did say, though, and this is why I am concerned, is that the brevetoxins could not have been the cause. I will just read from his statement where he says, "There was a lack of evidence to support that brevetoxin was the major cause of these strandings; the facts support an alternative conclusion that organochlorine compounds, in particular, polychlorinated biphenyls, PCBs, had an important role in the strandings."

In other words, what I am saying is—you tell me if I am wrong—everyone says that PCBs or contaminants may have played a role, and we need more study.

However, you say the brevetoxin was the trigger. Martineau says it couldn't have been. Others suggest other triggering mechanisms. For example, Dr. Vargo brought up the fact that if somehow the dolphins were starving, not because of brevetoxin, but for some reason they had to swim greater distances and didn't have access to food for some reason they would therefore ingest the blubber, and the PCBs would be a factor.

My concern is that the only point that everyone seems to agree on as a possible cause is the contaminants. Yet those are the very things that we don't seem to have the data or enough emphasis placed on them. So my conclusion from all that is let's do something. Let's reevaluate the data, if possible, to see if we can draw some conclusions about the contaminants and let's look into further possibilities in terms of correlation in that regard.

There is a definite discrepancy between your saying the brevetoxin is the triggering mechanism and some of the others saying it's not. I don't know how to clear that up. I would like to see more done with respect to the correlation, as you say, with these contaminants. That is my concern.

You certainly seem to agree that more needs to be done in that regard. I don't know how we are going to do it.

Mr. GERACI. Yes, indeed. You said everyone seems to agree—I didn't get that sense that everyone is agreeing, at least the scientific panel agreeing that contaminants are in fact much more important in this scheme. I know two of the panelists did agree.

That is not the issue. The issue is whether in fact we can make a correlation with scientific credibility linking the contaminant levels in the dolphins with specific effects as they might have been expressed in these animals.

I am suggesting to you that I cannot, and I don't know anyone who can.

Now, the burden of evidence is on me to support any statement I make, and if I made a statement going beyond those that I have made here today, I would not be able to support them. We are making a suggestion, and it's all we can do. Do studies need to be done? Of course. In the generic sense, we know nothing of the effects of contaminants on marine mammals.

So studies must be undertaken. If the questions is will further analysis of the tissues come closer to telling us why specifically the animals died? No, they won't. I can stand behind that with the burden of the evidence of the literature and my own studies on the actual event.

I don't believe that there is any supporting data in the literature which would suggest otherwise except for some plausible or possible links in one specific study in fact. So the large body of information on contaminant levels in dolphins, whales and dolphins, tells us nothing more than that they are there.

I can further add if I take all the rest of the tissues we didn't analyze from all 741 dolphins, every one of them would have contaminants in their blubber. We don't need to spend the money for the analysis. They have them. I can tell you that. But I cannot tell you what they do.

Mr. PALLONE. So is your answer to the question of whether or not analysis of the tissue that is available, be it frozen or whatever, in the lab situation, reevaluation will not help us in that regard?

Mr. GERACI. It will not.

Mr. PALLONE. Is there anything else then that this Committee could do, or that the investigation, or that NOAA could do to give us more clarity about the relationship of PCBs and other contaminants to dolphins? Again, going back to what I said initially yesterday, I am very concerned about what this means, not only for dolphins and other marine mammals but possibly for humans. You, yourself, do say very strongly, I think in the report you said, that there are high levels of—I used the term pollution, that these animals are swimming through and that that is a problem.

What can we do?

Mr. GERACI. There are indeed. I have asked the same question. It becomes a philosophical question. We are trying—what I see here is a very honest and legitimate, if you like, attempt to see just what these contaminants do. I would like to know what those contaminants do, if for no other reason that it finally ties together all this junk we are putting out on the health issue, because health issues are important. Health issues we deal with. Cosmetic issues, we don't. Even these issued we don't until it becomes a health issue.

If we cannot on the basis of this study make a precise determination that contaminants killed the dolphins and assuming that we reevaluate tissues for three or four years or for 10 and still fail to make a link, what do we do in the meantime; continue throwing contaminants in the ocean because we cannot link it to health? Or is it not enough to say we like our dolphins swimming in an ocean, that we are supposed to be protecting them, and we are not doing it.

Why do we have to wait until they get sick? Why do we have to wait for everyone to get lung cancer before we say stop smoking?

I want to see that ocean clean for them. It makes life a lot easier. Again, I cannot do it by tying this document to that goal.

Mr. PALLONE. All right. I appreciate that.

If I can just ask one more thing. Dr. Smayda made a point which I thought was a very good point. We talk about the red tide as a natural phenomena, yet he seemed to indicate it has become more frequent in the eastern waters of the Atlantic Coast, and more common and possibly more lethal, or more dangerous, and that it was linked to pollutants.

In other words, what I want to point out is even if I believe red tide was the cause, the bottom line is the red tide itself is becoming more frequent and that it is linked to pollution problems.

Could you comment on that?

Mr. GERACI. I would like to defer that question to Dr. Cross, if I may.

Mr. CROSS. I certainly agree with the comment made by Dr. Smayda yesterday, in general, that there seems to be an increasing abundance of those types of tides on a worldwide basis, and they are having quite a devastating effect on local ecosystems.

I think Dr. Smayda was talking in very general terms about what is happening globally. In the case of the red tide organism, we don't have any evidence at this time that its blooms are related to nutrient enriched waters. The blooms start offshore or on the outer edge of the shelf or Gulf Stream, or really, as Dr. Vargo stated, in nutrient poor or oligotrophic waters. But they always seem to initiate offshore where nutrients tend to be lower than in a coastal area, although I think for many types of phytoplankton the association of blooms with pollution is certainly the case. With this species, we have no link at this time to the massive blooms being tied to any pollution.

Mr. PALLONE. Thank you, Mr. Chairman.

Mr. FOGLIETTA. Thank you, Congressman Pallone.

Doctor, I am going to have to leave to go to another meeting. Before doing so, however, I wanted to thank you on behalf of this Subcommittee for a very thorough explanation and the answers to the questions were also very helpful. I want to thank you for the experts you have with you.

I would ask Congressman Pallone to take over the chair.

Mr. PALLONE. (Now presiding). Thank you, Mr. Chairman.

Mr. Carper.

Mr. CARPER. Thank you, Mr. Chairman.

Dr. Geraci, and to each of your panelists who join you today, we thank you for your presence. We thank you for your work on this effort and thank you for your willingness to share further in your ideas and to respond to our questions.

Let me just see if I have put together a clear understanding of the scenario that you have set forth. My understanding is, first of all, the dolphins ingested fish or something that contain brevetoxin and that brevetoxin then stressed the dolphins.

Second, the stressed animals apparently may have stopped eating and began to use their blubber in order to sustain themselves.

Third, the blubber contains contaminants. We talked about those contaminants, in some cases very high levels of contaminants. Those contaminants may or may not have further effect on the dolphin. Of that we are not sure, if it did, or how.

Finally, the seriously weakened animals succumbed to bacterial infections. Does that pretty well lay out what you have shared with us?

Mr. GERACI. Yes. That is fine.

Mr. CARPER. Do you think the animals would have died regardless of the contaminants they carried, or would the brevetoxin itself have sufficed to weaken the animals enough to lead to their demise?

Mr. GERACI. I think it is a two part question. Would they have died without the trigger from contaminants alone, is part of the question.

There is no evidence to support that. I don't know the answer. There is no evidence to support that.

Mr. CARPER. Say that one more time.

Mr. GERACI. There is no evidence that would support the probability of these dolphins dying, generically through the contaminant burdens because we have animals with high levels, animals with low levels. So, unless there is some trigger I can't put together a picture that would be plausible.

The other question, the other part of the question, would brevetoxin alone, without the contaminants—I don't know the answer to that.

Mr. CARPER. How do we find out the answer to that question?

Mr. GERACI. I guess directly we cannot, sir. But indirectly, there are lines of evidence with which we could come closer in years to answering that kind of question. Because we know a little bit about what brevetoxin does when it is metabolized, and goes to the liver to be processed for elimination, it utilizes the same pathways as do some of these contaminants. Contaminants need to be broken down as well to be excreted.

Well, the class of compounds are pretty similar, and they both need the same kind of machinery in the liver. It could be, I suppose theoretically, one could make—at least one could hypothesize that once an animal is putting all of his machinery at work processing contaminants, he may not have enough left over to process other biological toxins such as brevetoxin. So there is a hypothesis. It would have to be tested, of course, but I think it is reasonable. So, I think in a sense we can approach those questions indirectly.

Mr. CARPER. Can it be tested?

Mr. GERACI. That kind of study can be done. It is a laboratory study. Again, it is indirect and through that kind of study one does not say ultimately that therefore the contaminants killed the dolphins.

What one might say is that the contaminants will interfere with the processing of these biological toxins, and therefore, retard their exclusion, and therefore, encourage their presence in the mammal and make it more toxic. So that is indirect.

Mr. CARPER. You talked about the 44, I think you call them referees, who had reviewed the conclusions of your study and the conclusions regarding brevetoxin. We have heard from a couple of them yesterday apparently without our knowing it. Apparently some have agreed and some have disagreed with the conclusion.

Does the National Marine Fisheries Service have some idea of who has agreed, who had disagreed, and to what extent? Is that information available to them?

Mr. GERACI. Again, you didn't know it, but that was a two part question. Yes, the National Marine Fisheries Service does know, of course. They identified the referees. As part of the referee process, however, to protect the system, to allow us to have a system whereby we can produce creditable science and have it reviewed vigorously, the identity of referees is always protected, as is sometimes the process itself.

The National Science Foundation, for example, does not reveal, nor does a journal editor, reveal the summaries or the reviews. They are not made available to the public at large under any circumstances, very much like the relationships that lawyers hold with their clients. It is highly protected, and it is done that way deliberately to protect the system and to keep it as an integral part of the science.

Mr. CARPER. So what we will know then, given the way the system works, is that 44, at least 44 of your peers or colleagues reviewed our finding. We know of at least two who found disagreement with some of those findings, and we really won't know or have no way of knowing what conclusions the other 42 found.

Mr. GERACI. That, I don't know. I think you would have to go to the National Marine Fisheries Service for that information.

Mr. CARPER. Mr. Chairman, I would just hope we could ask for the record that the National Marine Fisheries Service make available to the Committee a summary of those conclusions and responses from the 44 referees. Is that a reasonable request?

Mr. GERACI. Not as far as I am concerned, but I think I have nothing to hide clearly. What I am trying to do is protect the process as I think you would try to project the client-lawyer relationship.

Mr. PALLONE. I am told by staff that the Chairman did make that request yesterday and that Dr. Evans has agreed to provide it without the names of the scientists, on an anonymous basis. So I guess we will get that.

Mr. CARPER. Dr. Geraci, in the legislation that we adopted last year in reauthorizing the American Animal Protection Act, we asked that the work that you were doing—I think we were aware you were doing that work at the time—we asked it be expanded as fully as possible to resolve a number of questions. One of those questions was the extent to which pollution may have contributed to dying. From what you have said, and the other witnesses have said, we know the pollutants were there at least in each of the animals that died, to one degree or the other. We don't know what those contaminants do. We simply don't know. How can we find out?

Mr. GERACI. It is going to be very difficult because we are working with marine animals. We know what those contaminants do in mice, rabbits, and laboratory animals, and in some cases domestic species, as well, because they are testable. But I do not want to be the person who submits a request for maintaining dolphins in captivity for the purpose of feeding them PCBs. So what we are left with is under the best circumstances extrapolating data from other species, dangerous unfortunately, especially with those compounds, because their effects are so varied.

Some animals need great quantities to produce any effect. Others less so with some animals the effect is on reproductive success. On others it might be the nervous system or the immune system. So one cannot readily extrapolate, at least cannot extrapolate with confidence, the data that come from studies on mice and others.

Unfortunately, I don't think we will ever be in a position—I wouldn't want to undertake the study to feed those classes of compounds to dolphins on a deliberate basis to determine what effect they have. So it is not going to be an easy one to solve.

Mr. CARPER. One last question, if I could. Someone else has already referred to it. Dr. Smayda raised the specter changes in global climate seem to precipitate an emotion of red tide and other kinds of blooms. In your hypothesis, biotoxins produced in these blooms might be the triggering mechanism for dioxin in marine mammals, is it fair to assume we might expect more of this kind of disaster in the future?

Mr. GERACI. Yes sir, I think it is. I think we just exposed a whole new line of thinking. I have been working with stranded marine mammals for many, many years and I have never examined any of them for biological toxins. I do it now. This past January a young hump back whale was stranded in Cape Cod. Under normal circumstances we would be taking our tissues and subjecting them to an analysis in the laboratory to see if we can find virus or bacteria. Now, we add saxitoxin to our repertoire and in that animal, we found it. I think many more animals have died than we detected because we have never analyzed for that toxin.

In the greater sense then, should the conditions, as rare as they seem to have been—that seemed to have prevailed—bringing together the dolphins, the fish that they ate, and the toxin, should they prevail again, there is no reason to assume we can't be facing or we won't face another mortality of this kind.

Mr. CARPER. Lastly, given the nature of your contractual relationship with the National Marine Fisheries Service, I presume your study is complete. Your conclusions are drawn and at this point in time do you walk away from this project and go on to others? Is this the end of the road?

Mr. GERACI. No, not at all. I think we need to discuss elements of this report, see where I might, if I can, help in any way to provide or to augment some of the data. I work with the National Marine Fisheries Service—I study natural mortality. I always work with them in stranding events. I don't see that I would walk away from this. I never have from a study I have been so intricately involved with.

Mr. CARPER. Again, thank you very much. Thank all of you.

Mr. PALLONE. Thank you.

Mr. Saxton.

Mr. SAXTON. Doctor, I hope you can understand we are trying a very hard to understand and reconcile differences of opinion that have to do with your report. I would like to point out five items that I find very difficult to reconcile. Perhaps you can help us reconcile them. Some of them have been talked about here today and yesterday, and some of them haven't. I asked a question relative to number one. If you could address each of these items when I am finished I would appreciate it.

Number one is, yesterday we had six of your colleagues here. When asked to respond to several questions, five of your colleagues responded and associated themselves with one statement that said they chose to associate themselves with a statement that said it is not reasonable to draw conclusions based on the data available in the study.

Second, your bibliography in your report makes reference to a study done by an R.H. Pierce in 1986. Apparently he is the Assistant Director of Moat Laboratory in Saratoga. I have not seen this study. Reportedly that study concluded that there is a "Negative correlation between red tides and dolphin deaths in the Gulf of Mexico."

Third, in the data in your report there is one dolphin which is identified as K-644, and he or she was found picked up off Cape Canaveral, Florida. That happens to be the dolphin carcass in which there was menhaden, which is identified as contaminated with brevetoxin. K-644 itself showed no evidence of brevetoxin.

Fourth, as we mentioned yesterday, the 17 dolphins which were studied for brevetoxin showed that eight of those dead dolphins actually had brevetoxin which was identifiable in their system, I believe. There was one manhaden which apparently showed evidence of brevetoxin, and that was apparently all that was found. So those are five points which have, to my satisfaction at least, not been reconciled.

The reason I think this answer is important is because the Members of this Committee—and the gentleman from Delaware in particular, when he sponsored the amendment which called for this study—were concerned about the ocean environment, were concerned about dolphins, but more than that, we are concerned about the ocean environment. To say that brevetoxin was the cause of these deaths in light of the fact that there seem to be what appear to me and I think other members of this panel, to be contradictions with your study, is difficult for us to reconcile that kind of thing.

Would you respond to those five points?

Mr. GERACI. Yes sir. I think they are very justifiable concerns. We have asked ourselves the same questions throughout the study. I think the first one is perhaps the easiest one for me to address.

The colleagues, the six respondents yesterday, none of them would have concluded in the same way, in other words, that they didn't agree with the conclusions. Agree. I don't have a conclusion. We are postulating a probable cause, not a conclusion.

So if the question had been posed another way, I wonder whether the answer might have been different. So, if we pose a question to a body of scientists to say "would you conclude in the same way"—first of all, I would hope the same information is available to the body of scientists. I felt they were put in an invidious position because they didn't have all the data that we had. They went on the basis of a single report.

As you will see when we submit the computer discs we have tens of thousands of data point of information. So they were put at some disadvantage in being asked the question. Nevertheless, they didn't conclude, nor can I conclude that that is why the dolphins died. We have probable cause and that is what we are suggesting.

Secondly, the Pierce report, I do not have the Pierce report, but we reviewed it. Dr. Pierce from Moat Laboratories, undertook a study, a retrospective study, to determine whether the incidence of brevetoxin organisms in the Florida Gulf where a population of dolphins reside, might be associated with increased strandings. Since we made a correlation on the East Coast they were wondering whether there may be a correlation on the West Coast, and he found none.

We know that in the West Coast—there are perhaps a number of reasons why that might be so. What is irrefutable, an incontrovertible fact, are the dolphins on the East Coast with toxic levels of brevetoxin in their livers. We don't have any data at all on brevetoxin levels in other stranded dolphins. So we can make one assumption—that the organism is on the West Coast all the time. If that is the case, and we have a background of strandings, why are they stranding? Maybe they are stranding because of brevetoxin. We don't know, because there has never been an analysis.

Secondly, we have reasonable evidence that the dolphin populations in the gulf reside in areas that are not typically bloom areas. They are in different regions. The other thing is—I think I will take a note from Congressman Pallone, who said yesterday these dolphins have some—they have got some pretty—they are pretty savvy creatures. They know their environment pretty well. They are only living in ten feet of water in that gulf and it is almost a two dimensional system.

We know the red tides are red. We know dolphins have very, very keen eyesight. We also know they can taste. We are pretty sure they can smell. A red tide is a pretty smelly place to be. They don't normally scavenge dead fish, so the fish that die with brevetoxin wouldn't be very attractive to a dolphin. So I don't know.

If I were a dolphin I don't think I would go in a brevetoxin contaminated area either.

Thirdly—and this is strictly hypothetical—if the dolphins have evolved in a system where there is brevetoxin persisting all the time, then they may have evolved some mechanism, behavioral avoidance perhaps, taste perception, some way to avoid it, and therefore, something that is there in their environment is something they can detect more easily. Whereas animals on the East Coast, not having had that advantage, might not be as aware.

The third point is dolphin K-644 that had brevetoxin in fish in its stomach but not in its liver. I think it is perfectly reasonable to assume that that animal might have been either feeding on toxin-containing fishes, which indeed we know it was, because it had the fish in its stomach, and had cleared other toxin before taking the fish.

We have no real problem with that. We have on the one hand, dolphins dead with brevetoxin in their liver. On the other, we have fish that they ate with brevetoxin in their livers. We know the dolphins ate the fish and I think we can make a pretty clear correlation between one and the other. Eight of the seventeen, not all, are a typical toxicology scenario. Some of the dolphins cleared the substance perhaps.

Other dolphins died with small and perhaps undetectable levels, and other dolphins we know in fact possibly died because they might have been abandoned by others or they might have—I shouldn't say that—young calves, for example, young animals which we had, may have been abandoned by their mothers, or perhaps their mothers died and they came ashore as a consequence.

So, not to find it 100 percent is actually good, because if you do, you start blaming the laboratory for contaminating the specimens. So I am actually pleased to see we hit on a percentage.

But we must not dismiss the fact it was 47 percent of the animals that died in association with the event. We found it in no cases of animals that were controls.

The last point I think is perhaps—I think I understand the question, but in fact we did not analyze one menhaden. We analyzed menhaden from the stomach of the dolphin. We also analyzed two lots of menhaden that were caught off Vero Beach in February, 1988 and from those two lots of fish that we caught, we got the toxin. So it was not a single fish.

Mr. SAXTON. Thank you, Mr. Chairman.

Mr. PALLONE. Thank you.

I am told we have to be out of here by about 1:25. I just wanted to ask a couple of questions and then if we have a few others that will be it.

The last question from Mr. Saxton made me think of a couple of other things. First of all, you suggested that perhaps the dolphins can avoid the brevetoxin. But my understanding was that the dolphins got sick from eating the menhaden. Now, they don't know when the menhaden are tainted by brevetoxin. There is no way they can determine that, right? It is not a question of them feeding on the brevetoxin. Is it not a question of them feeding on the menhaden that fed on the brevetoxin?

Mr. GERACI. Are we talking about the West Coast of the East Coast?

Mr. PALLONE. I am talking about dolphins on the East Coast.

Mr. GERACI. The dolphins on the East Coast would have died from the effects of ingesting fish containing brevetoxin, not the brevetoxin itself.

Mr. PALLONE. So I am saying there is no way for a dolphin to know not to eat certain menhaden because they have ingested brevetoxin. There is no way of their knowing that?

Mr. GERACI. I don't think so.

Mr. PALLONE. What about menhaden itself? Menhaden are caught as a species and made into fish meal, whatever. Are there any ramifications to humans from having eaten or had contact with menhaden that were infected by the brevetoxin?

Mr. GERACI. The menhaden that we tested were yellow fin menhaden which are not commercially exploited.

Mr. PALLONE. Not even for fish meal or agricultural purposes?

Mr. GERACI. I will defer the question to Dr. Cross, who is much more familiar with that than I, but in terms of—I can answer one part of the question. We have suggested that there be a monitoring program to determine whether brevetoxin is a component in plankton eating fishes, and furthermore, to determine what it actually does to the fish. Does it weaken the fish? What is the passage time through the fish?

So if there is a bloom, how long can we expect fish in that area to be contaminated? That information doesn't exist and we are suggesting that it should.

Dr. Cross can answer the part of that question.

Mr. CROSS. The yellowfin menhaden is not a very abundant species of menhaden as opposed, on the Atlantic Coast, to the Atlantic menhaden, which forms most of the catch. Until recently, some yellowfin menhaden were caught and processed.

Incidentally, they do commingle, in fact they even will hybridize with Atlantic menhaden off the coast of Florida. Two years ago the plant in Fernadina Beach closed, and there has been no active fishing for menhaden in Florida to my knowledge since that time. Occasionally yellowfin menhaden menhaden range from North Carolina to Georgia but to my knowledge, there is no active fishing of menhaden off the coast of Florida.

Mr. PALLONE. Doctor, I am not sure whether I understand what you are talking about as to how this report evolved. Yesterday I brought to the attention of Doctor Evans my criticism of the fact that at a press conference in February, when it was initially announced that the brevetoxin was the cause, my impression, having read that press release and the accounts that came afterwards, was that basically the announcement was that the dolphin deaths were caused by brevetoxin. Since then, until we received a report in April, which was a long time from the date of the press conference, when we read the report now it gives the impression that that is a hypothesis and other possible factors, such as PCBs and contaminants, may have come into play.

Are you suggesting that in the period between February and April the report was somehow changing because of the inputs of these 44 scientists? I wasn't clear how you said the report had changed or evolved over that period.

Mr. GERACI. Well, one is the issue of the press conference and the other is the report. The press conference was in the time frame available to make statements, we made very few qualified statements, but clearly, we were giving, again, plausible causes. And we had then evidence, the same evidence that we have now in fact on paper, that the contaminants were present in the dolphins and so on. So there was no difference. I don't think there is much inconsistency. There may have been inconsistency in the reporting but not in my impression, or as we have had those documents on paper.

The time lag that you are referring to is that between the submission of the final report and then the documents you see here. I don't know exactly how long it is, but it is probably two months or so, and I need some clarification about that. It took that long for, of course that number, that many reviewers to go through the paper, submit their findings to the National Marine Fisheries Service. They in turn, transmitted it to us, and we took the appropriate time to revise the paper based on the judgment of the referees.

Mr. PALLONE. In other words, these referees, or other scientists, their comments, criticisms, whatever, were incorporated into this final report to some extent?

Mr. GERACI. Yes. I think most of the changes, if I may—I think if they are made available to you, you will see—I think most of the changes were in the first cut we perhaps should have spoken more of plausible causes rather than—perhaps we were to conclusionary. That was a valid point, so we changed that.

Mr. PALLONE. I just wanted to ask two more things, perhaps somewhat parochial, that New Jersey people pointed out to me. One goes to Dr. Vargo's thesis that the dolphins might have to utilize more of that blubber and somehow the contaminants may have had an effect because of lack of food supply, or a different migration pattern.

I have been told that there wasn't much input from experts in the field of marine mammal behavior. Specifically, we had concerns that the Marine Mammal Stranding Center, which played some role in trying to rescue the dolphins, were not really consulted in a major way as part of this investigation.

Can you tell me why there wasn't more input from those in the field of marine mammal behavior? Because if I gave some credence to Dr. Vargo's thesis, or hypothesis, it would seem that migration patterns and the way dolphins travel, might be an important part of this picture.

Mr. GERACI. Yes. It would defy, I am afraid, our concept of why animals migrate in the first place. Dolphins won't go to the restaurant unless it is open. They went north because they were following food fish. And we have no indication at all the food fish were not as abundant that year as they were in previous years.

We did a host of behavioral studies. I sat there for hours and hours, sun up and sun down, watching dolphins to see what kind of clinical signs we might find. The only way we could do it was go where they fed every morning and every night. So there was lots of food there.

Mr. PALLONE. Did you have people in the field of marine mammal behavior involved in this investigation?

Mr. GERACI. No. We saw no need for people specifically dedicated to that. However, Dr. Ridgeway, who is probably the most notable neuro-biologist in our field, was part of our team. I know every marine mammal behaviorist in the country, and I think they would easily have come to provide aid had I requested them to do so.

To answer the second part of the question, the first week I arrived at Virginia Beach, I called Bob Shokoff and offered him the full services of the Department of Agriculture, told him we would bear the cost and we would send him, which we did, the containers to ship the tissues to the laboratory and we would provide as much logistic support as we could—which we offered, and he turned down—on a number of occasions.

We offered to send him people from our team, which he refused. We ultimately did send I think four or six samples to the Department of Agriculture, the results of which he has and he was free to send as many samples as he wanted.

I was in touch with Mr. Medway, his colleague, and colleague of mine for 25 years. I feel comfortable with the relationship we established. It wasn't a very close one. We had a lot of work to do and we offered to provide as much assistance as we could, but I couldn't force that on him.

Mr. PALLONE. The last thing is again a New Jersey question. In the back of the report a copy of the dolphins deaths is made available. I understand that even if the event didn't start in New Jersey, New Jersey had a lot of dolphin deaths take place in the state. Why was there such a high proportion of dolphins from southern states as opposed to New Jersey?

Mr. GERACI. Represented in—this is my study now? This is the study that begins with my having arrived in Virginia Beach. This is the result of my effort. These are the animals I can attest for, I will account and I will defend the results of. I can't do that with someone else's study.

It was my understanding 30 dolphins died in as many days in New Jersey. When I arrived, I asked for results or any help or tissues to send for analysis. There were very few. Some months ago, I called Dr. Rosco, who is a veterinarian, with the New Jersey Division of Fish, Game, and Wildlife, who did some of the necropsies and I asked him specifically whether he could provide tissues so we could actually detect brevetoxin in those tissues. He told me there were no tissues banked from those animals. I did what I could.

Mr. PALLONE. Any other questions?

Mr. SAXTON. I don't believe I have any other questions, Mr. Chairman. I just would like to say that if Dr. Geraci is right, and brevetoxin is the cause, that is scary, because what we have to do is sit tight and wait for another red tide and hope there is not a recurrence of the dolphin deaths.

If Dr. Geraci is wrong, then we look to toxics of other types as the proximate cause or probable cause of dolphin deaths. Then that is scary, too. The only thing we know for sure I think at this point, and the only conclusion that I can draw from the last two days of hearings, is that we have a scientific dispute between very well respected members of the scientific community as to what may have caused the dolphin deaths.

As you pointed out, Mr. Chairman, and as the gentleman from Delaware, Mr. Carper pointed out, I think what our position should be is we should throw the full weight of Congress behind further attempts to try and determine what the answers to this situation might be, and the causes of these dolphin deaths, and further, what other effect it may have or may be having, on the ocean environment.

Thank you.

Mr. PALLONE. Thank you.

I certainly agree, and I certainly appreciate Dr. Geraci's testimony today. I think I understand now fully what your thesis is, your hypothesis, and exactly what happened as a result of this investigation. But, of course, I also agree with Mr. Saxton that we really haven't gotten to the bottom of it. I hope through further efforts of this Subcommittee or Full Committee, that we can follow up in a sense on what you suggested, which is there need to be more study about the effects of contaminants and chemical pollutants and basically come to an answer.

But I appreciate your being here. Thank you all for testifying today. It certainly has been very informative.

Thank you.

(Whereupon, at 1:30 p.m., the Subcommittee was adjourned, subject to the call of the Chair.)

U.S. House of Representatives
Committee on
Merchant Marine and Fisheries
Room 1334, Longworth House Office Building
Washington, DC 20515-6230

MEMORANDUM

TO: MEMBERS, SUBCOMMITTEE ON OVERSIGHT AND INVESTIGATIONS

FROM: THOMAS M. FOGLIETTA, CHAIRMAN, SUBCOMMITTEE ON OVERSIGHT AND INVESTIGATIONS

DATE: MAY 8, 1989

RE: CONCLUSIONS OF THE CLINICAL INVESTIGATION OF THE 1987-88 MASS MORTALITY OF BOTTLENOSE DOLPHINS ALONG THE U.S. CENTRAL AND SOUTH ATLANTIC COAST

BACKGROUND

At 2:00 p.m. on Tuesday, May 9, 1989, in 1334 Longworth House Office Building, the Subcommittee on Oversight and Investigations will hold a hearing on the Conclusions of the Clinical Investigation of the 1987-88 mass mortality of bottlenose dolphins along the U.S. central and south Atlantic coast. Witnesses will include Undersecretary of Commerce for Oceans and Atmosphere, Dr. William Evans, the principal investigator of the mass mortality study, Dr. J.R. Geraci, and a panel of six marine scientists who will critique the methodology and findings of the eighteen month clinical investigation.

HISTORY

In late June 1987, the first of the dead or dying Atlantic bottle-nosed dolphins washed ashore in southern New Jersey. As the summer progressed, dolphins began stranding down the coast into the Carolinas. Eleven months after the first stranding, the last of 742 dolphins washed up on Florida's east coast. It is estimated that several thousand others also died but did not wash ashore.

Late fall 1987 also saw thirteen humpback whales wash ashore on Cape Cod. These marine mammal deaths coincided with many beach closures in the northeast in 1987, primarily in New

Jersey, where human wastes, garbage and medical wastes washed up on shore. The 106 Mile Dump Site was also opened for sewage sludge disposal on March 17, 1987.

In late June 1987, the Marine Mammal Stranding Center in Brigantine, N.J., began an investigation of the dolphin strandings. In August, 1987, as the strandings continued to increase in number, an investigative task force was formed by the U.S. Marine Mammal Commission and the National Oceanic and Atmospheric Administration (NOAA) to determine the cause. The investigation was led by Joseph R. Geraci, V.M.D., Ph.D., of the Ontario Veterinary College, a world renowned expert on marine mammal diseases. Dr. Geraci's first task was to set up a team to do the investigation. This was being done at a time when public speculation and rumors were running rampant about contagious diseases, such as AIDS, being responsible for the deaths.

Tips and theories on the dolphin deaths were so numerous that Dr. Geraci's team, at one point, reached 30 people. (Temporarily, the team even included a blue crab specialist from the University of Maryland to examine a link between crabs washing ashore and the dolphin deaths. The crab strandings turned out to be the routine result of lower dissolved oxygen levels due to the hot summer.) The team worked out of Virginia Beach, Virginia.

The dolphin deaths, however, were far from a routine occurrence. In a typical year, there are fewer than recorded 50 dolphin deaths along the Atlantic coast. Although no census has ever been taken, the East Coast population of bottle-nosed dolphins prior to the die off was believed to be approximately 10,000--one-half of which are the near-shore migratory stock. Estimates are that 2,500 dolphins, or fifty percent of this near-shore coastal migratory stock, died during the epidemic. (The near-shore coastal migratory stock are those animals that migrate no more than 100 miles from shore.) The separate dolphin population that migrates more than 100 miles from shore apparently was unaffected by the epidemic.

The Geraci team spent eighteen months before finalizing their report in February, 1989. The team obtained data or specimens from 347 of the dead dolphins, and blood samples were taken from 23 live animals that were captured off Virginia Beach in August and October, 1987. Samples from freshly dead animals were used to study pathology, virology, microbiology, and chemical and biological toxicology. The blood samples were analyzed for hematology, proteins and protein electrophoretic patterns, thyroid and adrenocortical hormones, and viral antibodies.

INTERIM REPORT

On May 3, 1988, the Geraci team submitted an interim report to the U.S. Marine Mammal Commission. The Geraci team indicated that they were pursuing four areas of investigation which may have been the cause of the dolphin deaths: 1) bacteriological; 2) virology, including some type of "dolphin AIDS"; 3) environmental correlates, including the effects of sewage sludge dumping at the 12 Mile and 106 Mile Dump Sites, and high levels of heavy metals and contaminants; and 4) toxicology, which was enhanced by the finding that some Atlantic mackerel taken from the stomach of one of the humpback whales stranded on Cape Cod tested positive for paralytic shellfish poisoning.

MARINE MAMMAL PROTECTION ACT AMENDMENTS OF 1988

On November 23, 1988, the Marine Mammal Protection Act Amendments of 1988 (P.L. 100-711) was signed into law. The law contained a provision (Section 7(a)) offered by Rep. Thomas R. Carper to require a study by the Department of Commerce into the dolphin deaths. Specifically the provision required an examination of:

"1) the cause or causes of the epidemic;

2) the effect of the epidemic on coastal and offshore populations of Atlantic bottle-nosed dolphin;

3) the extent to which pollution may have contributed to the epidemic;

4) whether other species and populations of marine mammals were affected by those factors which contributed to the epidemic; and

5) any other matters pertaining to the causes and effects of the epidemic."

In a letter dated December 30, 1988, the Department of Commerce advised both the Committee on Merchant Marine and Fisheries and the Committee on Commerce, Science, and Transportation of the Senate that the Geraci study would fulfill all of the requirements of Section 7(a). [Letter attached.] Dr. Geraci revealed the preliminary results of the study at a press conference on February 1, 1989, and the final report became available to the Committee on Merchant Marine and Fisheries on April 26th.

CONCLUSION OF THE GERACI INVESTIGATION

Among the findings of the Geraci study was that, "this has been the most extraordinary saga of cetacean disease on record." This appears to be the only major aspect of the investigation that is agreed upon by all of the experts in the field.

The Geraci investigation concludes that the dolphins died due to ingesting fish tainted by the naturally occurring toxin, "brevetoxin," produced by "red tide algae." The scenario established by the Geraci study is that a "red tide" bloomed in the Gulf of Mexico in February, 1987. A portion of the algae bloom drifted to the east coast of Florida by the fall. As the dolphins made their annual northern migration during the spring, they fed on fish (i.e., menhaden, mackerel) that had been contaminated by the brevetoxin from the red tide. The Geraci investigation concludes that most of the dolphins had their immune systems severely weakened by the brevetoxin, and that their actual causes of death were various infections and diseases.

The investigation concludes further that in the fall of 1987, on their southernly migration, the dolphins encountered the same bloom; by then it had migrated north to the coast of North Carolina. This second encounter was responsible for the wave of stranded dolphins along the Florida coast in the winter of 1987-1988.

The investigation also finds that there were high levels of toxins, such as PCBs and DDEs (a derivative of DDT), in various organs of the dead dolphins. Dr. Geraci, however, concludes these high levels of contaminants were not the "key" to this event.

ISSUES

Requirements of Section 7(a) of P.L. 100-711. The study by Dr. Geraci was well under way when Rep. Carper introduced his amendment to the Marine Mammal Protection Act Reauthorization. On December 30, 1988, the Department of Commerce notified the Committee that the Geraci study would fulfill all of the requirements of Section 7(a). Nonetheless, there is concern that it does not adequately address several specific points:

* Section 7(a) requires NOAA to look at "the effect of the epidemic on coastal and offshore populations of Atlantic bottle-nosed dolphin." The study estimates that 50% or more of the coastal migratory stock died during this period. However, it offers no estimates as to the size of the stock, nor does it report on any effects to the offshore population.

* Section 7(a) requires NOAA to look at "the extent to which pollution may have contributed to the epidemic."

The preliminary study released in May, 1988, discussed the need to acquire data on the types and amounts of industrial and municipal wastes discharged into mid-Atlantic coastal waters--specifically data concerning the 12 Mile and 106 Mile Dump Sites. No such data, nor any conclusions about it were mentioned in the final study.

If the requirements of Section 7(a) have not been met in the Geraci study, where and when will they be addressed by NOAA?

Environmental Causes. Specifically,

* Was pollution in any way responsible for the unusually large and long lasting red tide algae blooms?

* What was responsible for the remarkably high levels of contaminants in the dead dolphins?

* Did the opening of the 106 Mile Dump Site on March 17, 1987 and the problems of combined sewer overflows and floatables that plagued the beaches during the summer of 1987 play any role in the dolphin deaths?

Methodology. A number of questions have arisen pertaining to the reliability of data and adequacy of sample sizes for drawing conclusions. For example,

* The conclusion that the dolphin deaths were caused by brevetoxin was based on the positive test results from eight dolphins out of a total test sample of 17. Is this sampling conclusive? (A total of 744 dolphins washed ashore.)

* Was the quality of the tissue and organ samples used in the study adequate? Generally, the fresher the sample the better. In some cases, tests were run on frozen tissue and organ samples. Moreover, often it was impossible to know how long an animal had been dead before washing ashore.

* Is there a proven link between the lesions found on the dead dolphins and brevetoxin? None is apparent in current literature. Was data which links lesions to organochlorine compounds in laboratory and domestic mammals ignored?

Peer Review Process. Apparently, the peer review comments on the investigation's finding were highly critical. Therefore,

* Were the criticisms expressed in the peer review process incorporated in any way into the final report?

Red Tide. Red tides are a naturally occurring phenomena and are common in the Gulf of Mexico where they appear to coexist with dolphins. Therefore,

* If a naturally occurring red tide was responsible for the dolphin deaths, why did it happen in 1987, but never before?

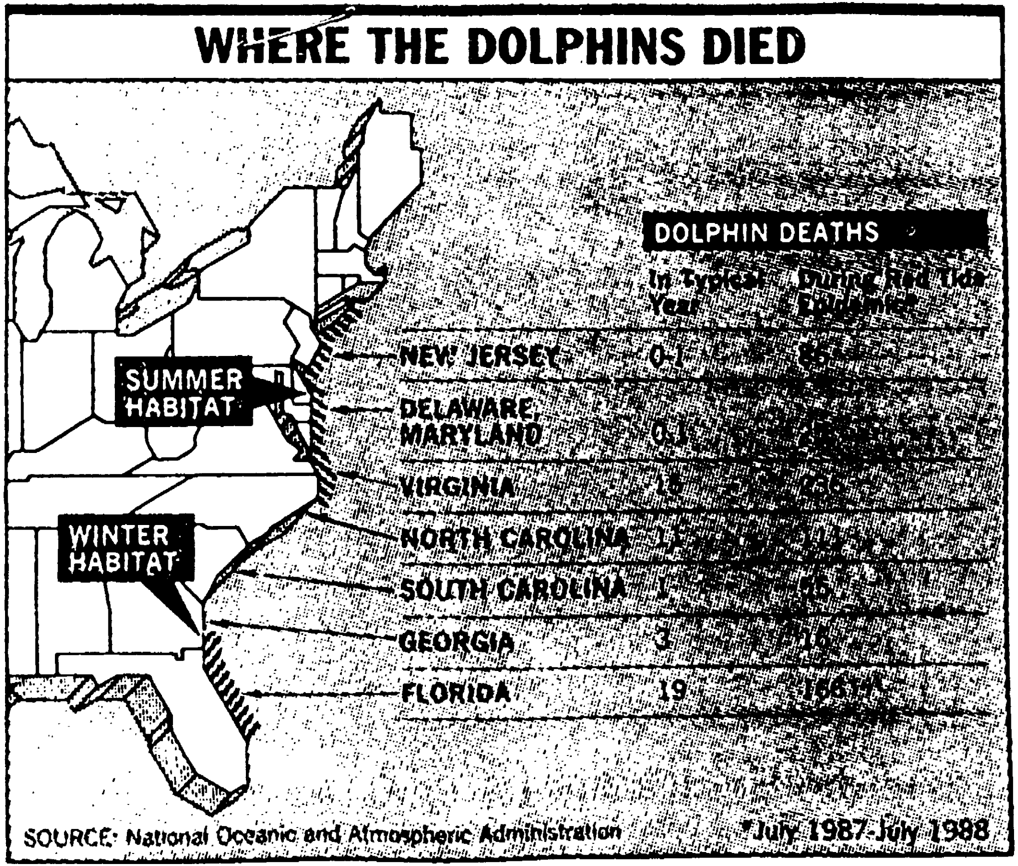
* What could have caused the unusual red tide to last for eight months and to move from the Gulf of Mexico to the coast of North Carolina?

* What are the chances of a reoccurrence of this situation, and what can be done to prevent it?

* Does the red tide's appearance coincide with the migratory patterns of the dolphins and the "contaminated" menhaven fish the dolphins feed upon?

Contaminants. The investigation has been criticized for ignoring excessively high levels of contaminants in the dead dolphins, specifically PCBs and DDE. The PCB level in one mature male specimen was 6,800 parts per million. (The Food and Drug Administration's acceptable level for human consumption of food is 2 ppm.)

* Why was this information dismissed as having no effects on the dolphin deaths?



BY BRAD WYE—THE WASHINGTON POST

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May 5, 1989

TO : House Committee on Merchant Marine and Fisheries
Subcommittee on Oversight and Investigations
Attention: Phillip Rotondi

FROM : Eugene H. Buck EHB
Specialist in Natural Resources Policy
Environment and Natural Resources Policy Division

SUBJECT : Comments on Dolphin Mortality Final Report

In response to your request that CRS review and comment on the final report entitled "Clinical Investigation of the 1987-88 Mass Mortality of Bottlenose Dolphins along the U.S. Central and South Atlantic Coast," I have reviewed this document and offer the following summary comments:

Background

1) The study was quite comprehensive in its scope of possible factors evaluated for their potential contribution to the dolphin mortalities. Methods generally appeared to be state-of-the-art and appropriate to the situation.

2) Notably lacking were organized discussions of a) the areas and times of documented dolphin mortalities, and b) the areas and times of *Ptychodiscus brevis* blooms which could have contributed to the mortalities. Without these data presented in a logical, organized manner, it is difficult to evaluate conclusions concerning the plausibility that these blooms were prime contributing forces to the mortalities. The discussion (p. 18) of bloom occurrences is not well organized in its presentation, and should be more extensive and appear earlier in the document to provide background information. This problem is perhaps understandable in a document whose parts were contributed by so many investigators; however, more effort might have been taken to present essential background information in a well-organized manner.

CRS-2

Data

1) I was unable to readily identify if adequate controls were used so that the results could be compared in order to understand how typical/atypical the observed physiological/pathological conditions might be in the bottlenose dolphin population. If appropriate controls were not available, this fact should have been stated clearly to set the findings in proper perspective. This was a particular deficiency with the pathology (p. 9) and bacteriology (p. 10) sections, but persisted in other discussions as well. Without controls, one cannot clearly evaluate to what degree the observed conditions possibly contributed to the mortalities.

2) The several species of fish tested for brevetoxin (p. 8) were not identified as being selected specifically for their predominance in the diet of bottlenose dolphin or for their association with areas affected (at the time of fish capture) with *Ptychodiscus brevis* blooms. Assurances of connections between collected data and behavioral/environmental factors necessary to substantiate probable causes are essential to evaluate the conclusions.

Interpretation

1) Information presented on p. 16 establishes the fact that the dead dolphins inhabited a very polluted environment. I don't feel sufficient consideration was given to the possibility that poor habitat weakened the dolphins, making them susceptible to bacterial invasion (p. 9) and immunoincompetence (p. 15), eventually resulting in the observed massive mortalities.

2) Insufficient evidence is presented to support the conclusion that brevetoxin contributed significantly to mortalities prior to Sept.-Oct. 1987. If information available on dolphin mortalities and *Ptychodiscus brevis* bloom time and space relationships had been better presented (see Background - item 2, above), this conclusion might be strengthened.

3) There is little discussion of how the observed symptoms might have been produced in animals exposed to brevetoxin. In fact, discussion of symptoms related to polychlorinated biphenyl (PCB) toxicity (p. 16) superficially appeared to be closer to the observed symptoms. I am not implying that PCBs were the cause of the observed mortalities; only that, in the absence of observed symptoms which can be related directly to brevetoxin, alternative explanations can be just as, or more, plausible.

4) The reliance on theoretical sublethal effects of brevetoxin exposure (p. 18) as weakening the dolphins is not well supported by direct observations, either connected with this event or observations cited from other sources. It is a hypothesis which is difficult or impossible to test and evaluate. Therefore, I don't find this argument persuasive.

5) Even if one accepted the possibility that sublethal effects weakened the dolphins (see item 4, above), why might not exposure to the morbillivirus resembling canine distemper virus (CDV) (p. 15) be just as plausible a cause of death as brevetoxin, since the occurrence of CDV-type antibodies appears just as prevalent as brevetoxin in the dead dolphins? Or even poor habitat quality (see Interpretation - item 1, above) acting to weaken dolphins?

6) Since the report states "systemic bacterial invasion ... seems to have been ultimate cause of death of many of the dolphins" (p. 9), and "the overwhelming nature of some of the infections, which probably arose in the lung, may have been related to immunoincompetence, the cause of which cannot be established" (p. 15), there appear to exist equally plausible alternative conclusions suggested that mortalities were related to situations not associated with *Ptychodiscus brevis* blooms. In fact for early mortalities (see Interpretation - item 2, above), alternative explanations appear more plausible.

Although dolphins undoubtedly die from brevetoxin, I do not find the evidence compelling that brevetoxin caused or contributed to the preponderance of mortalities. The evidence may exist to build a better case for this causal relationship, but the lack of evidence presented in the final report does not seem to justify an unequivocal determination.

I can be contacted directly at 7-7262 if you have questions on this critique, or should you require additional information or analysis on this or a related subject.

UNITED STATES DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
NATIONAL MARINE FISHERIES SERVICE
Silver Spring, Maryland 20910

DEC 30 1988

Honorable Walter B. Jones
Chairman, Committee on Merchant
Marine and Fisheries
House of Representatives
Washington, D.C. 20515

Dear Mr. Chairman:

Section 117 of the Marine Mammal Protection Act Amendments of 1988 addresses the requirement for a study regarding the east coast epidemic and subsequent mortality of the North Atlantic coastal population for the bottlenose dolphin. It requires the Secretary of Commerce to submit to the Senate Committee on Commerce, Science, and Transportation and the House Committee on Merchant Marine and Fisheries, by January 1, 1989, a plan for conducting the study.

The work on the 1987 dolphin die-off has been an ongoing effort involving an unprecedented level of cooperation among agencies, private and public organizations and individuals. The investigation is being directed from, and the results collated and interpreted at, the Ontario Veterinary College, under the direction of Joseph R. Geraci, V.M.D., Ph.D. Dr. Geraci is presently under contract with our agency. The study has received support from the Office of Naval Research and the Marine Mammal Commission as well. Enclosed is a copy of our Cooperative Agreement with Dr. Geraci which outlines the plan for carrying out this work as required by the amended legislation.

The investigation is essentially completed and we expect a final report by January 30, 1989. Within 60 days of our receipt, the Secretary will forward a copy to you. The report will contain a discussion of the causes and impacts of the 1987 epidemic. It will also address the questions contained in Section 117 of the amended Marine Mammal Protection Act and describe any follow-up actions we feel should be taken.

We appreciate your interest in this study.

Sincerely,

James W. Brennan

James W. Brennan
Assistant Administrator
for Fisheries

Enclosure

NOAA

THOMAS R. CARPER
DELAWARE, AT-LARGE

COMMITTEES:
BANKING, FINANCE AND URBAN AFFAIRS
MERCHANT MARINE AND FISHERIES

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House of Representatives
Washington, DC 20515

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May 1, 1989

Mr. James W. Brennan
Assistant Administrator for Fisheries
National Marine Fisheries Service
National Oceanic and Atmospheric Administration
1335 East West Highway
Silver Spring, Maryland 20910

Dear Mr. Brennan:

I am writing in response to the report written for the National Marine Fisheries Service (NMFS) by Dr. J.R. Geraci titled "Clinical Investigation of the 1987-1988 Mass Mortality of Bottlenose Dolphins Along the U.S. Central and South Atlantic Coast." I believe this report raises serious questions about what role pollution in our coastal waters may have played in the die-off.

During last year's consideration of the reauthorization of the Marine Mammal Protection Act, I authored an amendment to require the NMFS to investigate — 1) the cause or causes of the dolphin die-off; 2) the effect of the die-off on inshore and offshore populations of east coast Atlantic bottlenose dolphins; 3) to what extent pollution may have contributed to the die-off; 4) whether other species of marine mammals were affected by those factors which caused the Atlantic bottlenose dolphin die-off; and 5) any other matters regarding the causes and effects of the die-off. I am extremely concerned about the possibility — noted by Dr. Geraci in his report — that contaminant levels found in the dolphins could have contributed to this massive loss of coastal bottlenose dolphins.

Since the study I added to the Marine Mammal Protection Act bill passed last year requires an exhaustive investigation of, among other things, "to what extent pollution may have contributed to the die-off," I suggest that NMFS continues that part of the study I requested so that we might better understand the impact of coastal pollutants on marine mammals living in those areas. Consistent with the timetable established by my amendment, I would hope that the NMFS could respond to the Committee on Merchant Marine and Fisheries, of which I am a member, and to the Senate Committee on Commerce, Science, and Transportation on or before January 1, 1990, on what continuing activities NMFS will undertake to further resolve these remaining, critical questions.

Mr. James W. Brennan
May 1, 1989
Page two

It is essential that we develop a clear understanding of our impacts on coastal waters and their inhabitants if we are to take appropriate actions to protect them. I will gladly work with you and your colleagues at the National Marine Fisheries Service to accomplish this important goal. I appreciate your interest in this issue, and hope you will contact me if there is anything more I, or Congress, can do to assist you.

Sincerely,

Tom Carper

Tom Carper
Member of Congress

TRC/ct

MOTE MARINE LABORATORY

1600 CITY ISLAND PARK
SARASOTA, FLORIDA 34236
PHONE (813) 388-4441

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RED TIDE AND DOLPHIN STRANDINGS IN THE GULF OF MEXICO

Richard H. Pierce, Ph.D.

A comparison between reported dolphin strandings and red tide blooms from data collected in the coastal Gulf of Mexico (from Texas to Florida 1985 through 1987) showed no relationship of dolphin strandings to red tides. Red tide data were obtained from records of the Fl. Dept. of Natural Resources and Mote Marine Laboratory. Dolphin strandings were obtained from reports of sitings compiled by the Mote Marine Laboratory Marine Mammal Program.

Specific observations from these data include:

1) Red tides occurred primarily along the southwest coast of Florida, whereas dolphin strandings were reported throughout the Gulf of Mexico Coastal region.

2) More dolphin strandings were reported from areas where red tides normally do not occur than areas where red tides routinely occur.

3) Dolphin strandings reported during and after a severe red tide bloom along the Texas coast in Aug.-Oct., 1986, were less than the number reported earlier in the year for the same region.

4) No correlation was found between the incidence of red tide blooms and dolphin strandings along the southwest coast of Florida for the two most recent years for which complete data sets were available; 1986 (cor. coef. = 0.14) and 1987 (cor. coef. = -0.23)

Conclusion: In the Gulf of Mexico, there is no correlation between reported dolphin strandings and observed incidences of red tide, indicating that, in this region, red tide is not a major factor in dolphin strandings.

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MOTE MARINE LABORATORY RED TIDE RESEARCH PROGRAM

Richard H. Pierce Ph.D.
Associate Director and Senior Scientist

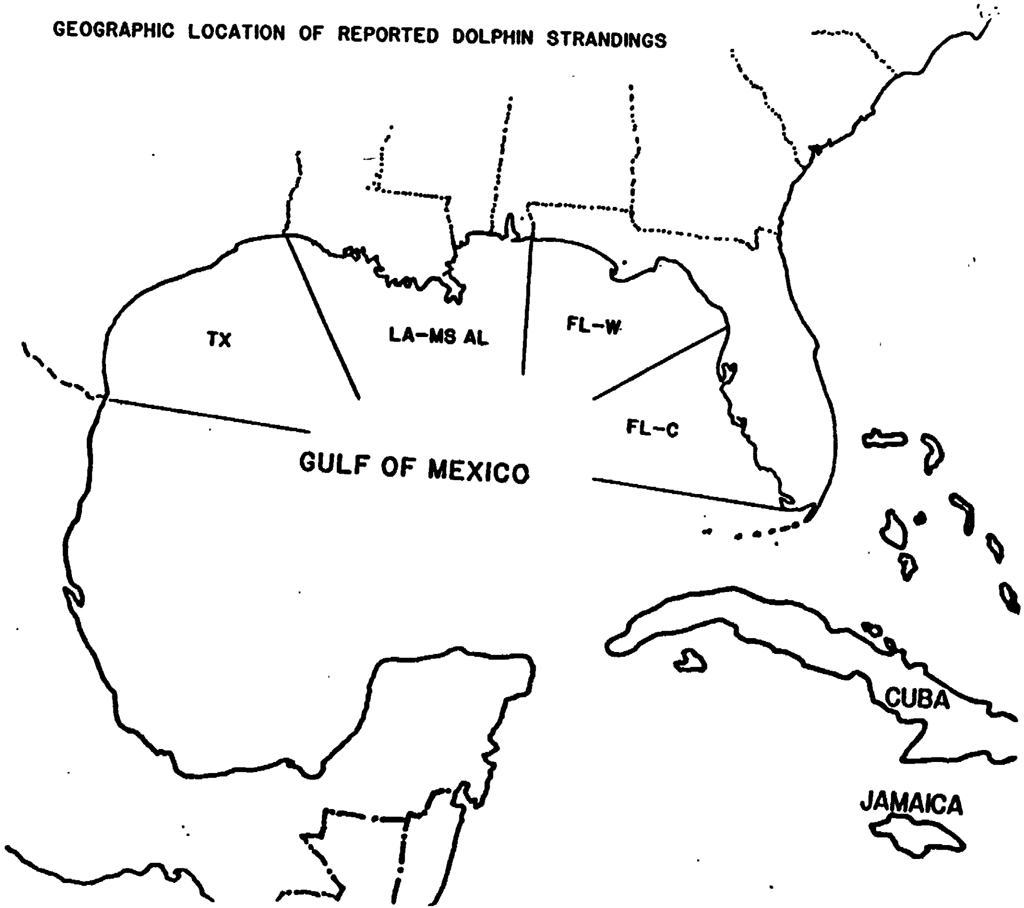
Red tides occur worldwide resulting from natural blooms of phytoplankton. The Florida red tide is caused by periodic blooms of the dinoflagellate, *Ptychodiscus brevis*. This microscopic marine alga produces several chemical toxins that are released to the water when the cell membrane ruptures, causing massive fish kills, contaminating shellfish, and causing severe respiratory irritation when blown ashore with marine aerosols.

The Mote Marine Laboratory (MML) Red Tide Research Program is carried out in cooperation with the Florida Department of Natural Resources, Florida Marine Research Institute (DNR-MRI). The MML program focuses on toxins produced by *P. brevis*, and the effects of these toxins on aquatic organisms as well as on humans. Laboratory cultures of the red tide organism are maintained at MML for carefully controlled laboratory studies, and to support field investigations of red tide blooms.

Current investigations address the potency of *P. brevis* toxins to different life stages of fish to help understand how red tides affect fish populations along the Florida Gulf Coast. Considerable effort also is directed toward studies of the production and transport of airborne toxins (aerosols) which affect the human respiratory system, as well as marine bacteria which are associated with red tides and may also be incorporated with marine aerosol that is impacting humans. In addition, MML continues to identify and monitor red tide blooms as a service to the State for public health considerations. MML scientists alert the DNR to red tides in the Sarasota and Manatee County areas and provide updates of cell population counts as well as monitoring movement of the red tide blooms, to identify impact of new areas or removal from affected locations. Future studies will assess bioaccumulation of toxin in fish exposed to sublethal concentrations of red tide. This information is essential to understanding such critical problems as the deaths of hundreds of marine mammals along the Atlantic coast.

The overall goal of the Mote Marine Laboratory Red Tide Research Program is to provide a better understanding of this natural event, specifically concerning the chemical toxins produced and their effects on humans as well as marine organisms. MML is cooperating with various state and federal agencies to gain new knowledge about red tides and to evaluate the potential for alleviating the adverse effects without inflicting ecological damage.

GEOGRAPHIC LOCATION OF REPORTED DOLPHIN STRANDINGS



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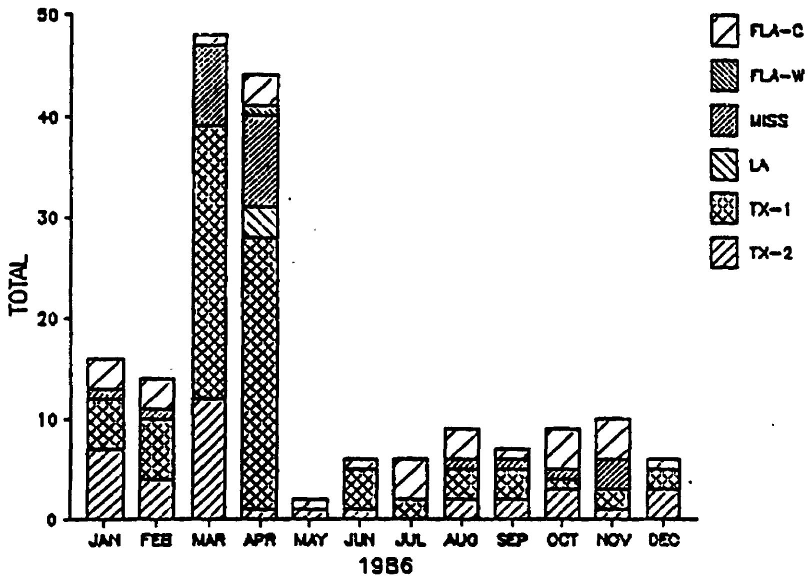
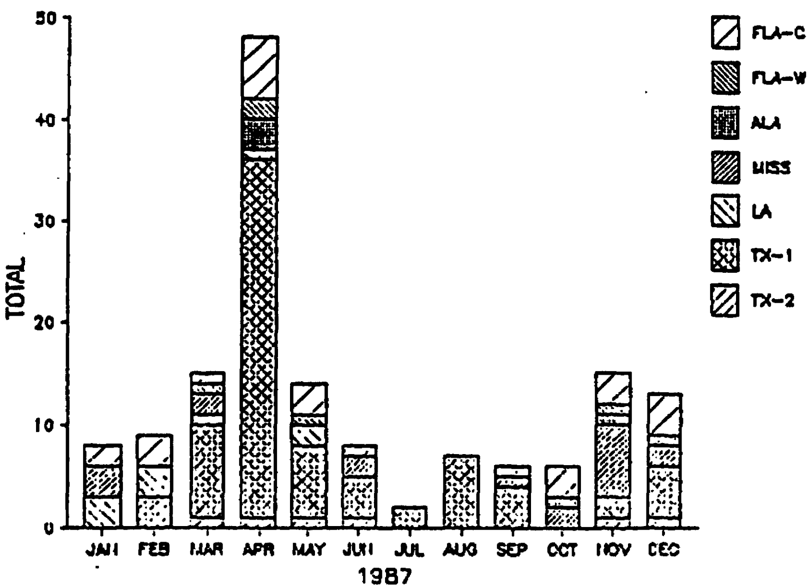
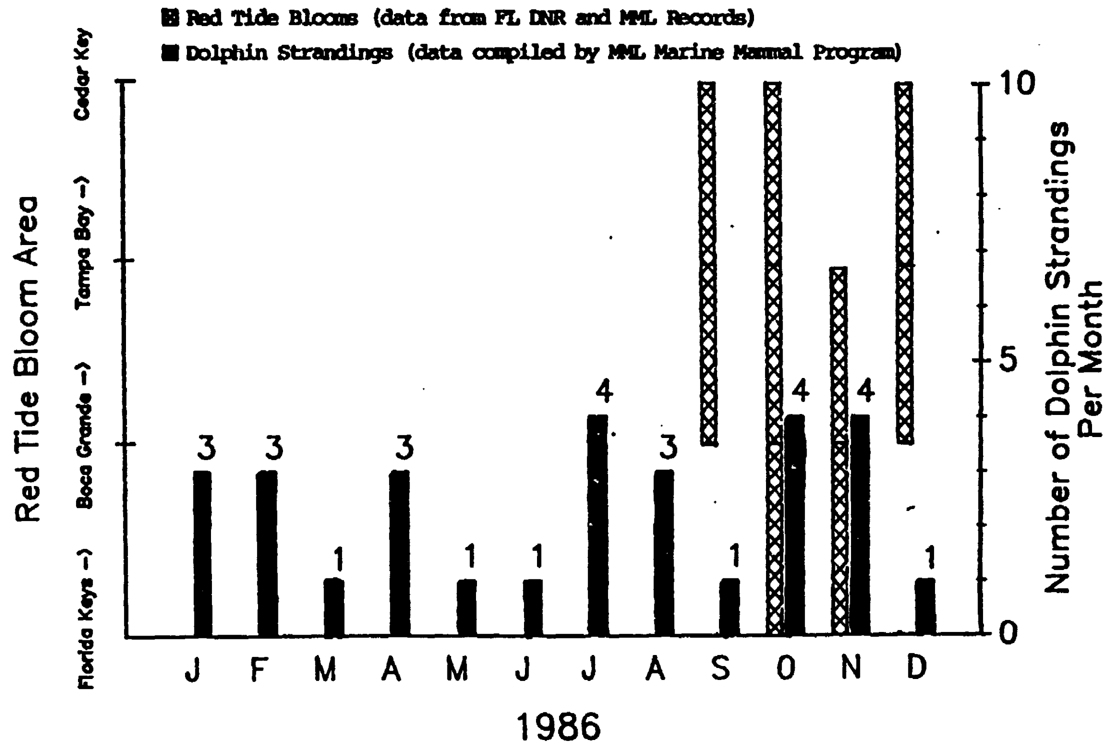
REPORTED DOLPHIN STRANDINGS

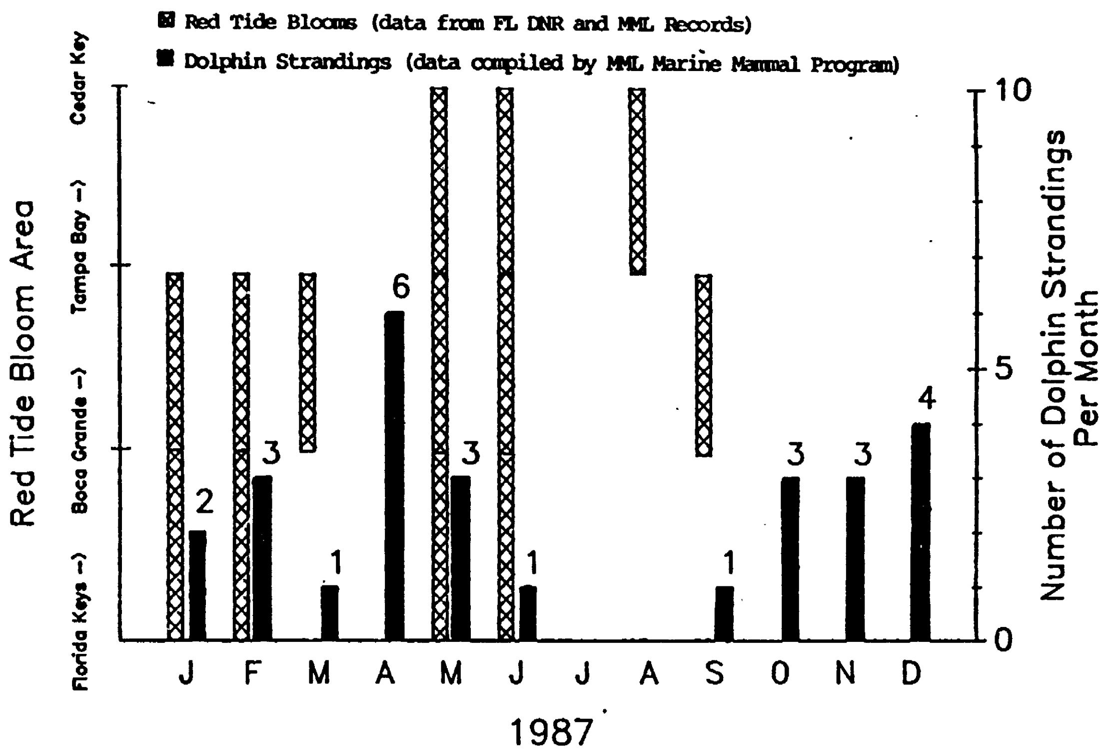




COMPARISON OF RED TIDE BLOOMS WITH DOLPHIN STRANDINGS ALONG THE SOUTHWEST FLORIDA GULF COAST, 1986. (Cor. Coef. = 0.14)



COMPARISON OF RED TIDE BLOOMS WITH DOLPHIN STRANDINGS ALONG THE SOUTHWEST FLORIDA GULF COAST, 1987. (Cor. Coef. = -0.23)



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April 30, 1989
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Joseph E. Cummins

Comments on "Investigation of the 1987-88 Mass Mortality of Bottlenose Dolphins along the U.S. Central and South Atlantic Coast" by J.R. Geraci, January 1989 a final report.

Comments By: J. B. Cummins
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BEST AVAILABLE COPY

This report studied pollution in bottlenose dolphins who died in great numbers along the Atlantic coast of the United States during 1987-88. The report concludes that the dolphins were poisoned by brevetoxin, a product of a red tide organism. The animals also suffered monumental pollution with PCBs and other organochlorine pollutants and suffered massive immune suppression finally causing most to expire from chance infection.

First, the brevetoxin hypothesis was not entirely convincing for two reasons: the first reason is that about half of the dead animals showed little or no evidence of the toxin while all of the dead animals showed high levels of pollution with PCBs and mercury; the second reason is that the animals showed extensive immunotoxicity while brevetoxin is a neurotoxin but has not been reported to be immunotoxic (Envir. Health Criteria 37 Aquatic Biotoxins, World Health Organization 1984). Current reviews of immunotoxic agents list PCBs and mercury but none of these report brevetoxin as an immunotoxin. The author of the current report should be required to provide data supporting the assumed immunotoxicity of brevetoxin before assuming it to be the case.

Geraci has unusual ideas about PCBs among which is the comment (page 20) "the mere presence of organochlorines in blubber poses no apparent risk." As a rule serum levels of halogenated biphenyls have been found to be strongly related to levels in adipose tissue (i.e. Kreiss, et. al. Arch. Envir. Health 37, 141, 1982). Geraci did not provide scientific support for what seems to be a misunderstanding on his part. Similarly, high liver levels of PCBs

2

such as those observed by Geraci can be caused by contemporary sources of PCBs pollution (i.e. ocean dumping) or by cachexia (vasting disease). The contemporary source hypothesis is consistent with the reported state of the animals, none of which are reported to be cachexic.

• Geraci seems to have fudged his PCBs analysis so as not to "skew" the population mean (page 14). The observed PCB level in blubber 6800ppm (0.68%) and 5200ppm in liver (0.52%) is the highest PCB level yet reported in any animal to the best knowledge of the reviewer. The PCB levels reported in Atlantic bottlenose dolphins were on the average about five times greater than the PCBs observed in white-beaked dolphins trapped in ice near Newfoundland (Muir et. al. Arch. Envir. Cont. Tox. 17, 613, 1988). The bottlenose PCBs were 20 to 30 times greater than values reported for North Pacific Dalls porpoise (Subramanian et. al. Marine. Envir. Res. 25, 161, 1988). It seems likely that the elevated levels of PCBs observed by Geraci were sufficient to compromise the immune systems of the beached animals. However, direct experimentation would finally establish whether or not PCBs were the main cause. Nevertheless a comparison with laboratory animals studies or human studies support the hypothesis (Chang et. al. J. Tox. Envir. Health 9, 217, 1982).

Levels of mercury observed by Geraci were significantly greater than the mercury values observed by Muir et. al. (Arch. Envir. Contam. Tox. 17, 613, 1988). Geraci did not find this pollution noteworthy.

3

Even though lymphoid follicles were depleted in spleen, lymph nodes and intestine observations were not reported for the pathology and size of the thymus (the primary organ of the immune system). Tumors were not reported in any of the organs of any of the animals. This is a surprising finding but it was not discussed by Geraci.

In conclusion, Geraci concluded that brevetoxin poisoned bottlenose dolphins along the Atlantic Coast. His evidence indicated that the animals had elevated exposure to PCBs most likely of contemporary origin (i.e. ocean dumping) but Geraci did not believe that the PCBs posed a problem.

SICK SEAS: 1988 saw a mass epidemic of seal disease in the North Sea. Pollution has drastically weakened their immune system and there are real fears for the future survival of the species.

Extinction:
The PCB Threat to Marine Mammals

by
Joseph E. Cummins

Within the decade, most of PCBs now in use will wear out in both industrial and developing countries. Few Third World countries have the funds or the political will to ensure their proper disposal. Yet if the PCBs held in the Third World alone were released into the general environment, the extinction of marine mammals would be inevitable. To avoid disaster, PCB manufacturers should "buy back" their products and pay for their safe disposal.

Recent studies have detected a very alarming trend in the accumulation of polychlorinated biphenyls (PCBs) in the waters of the oceans and their biomagnification to elevated levels in the tissues of such marine mammals as whales, dolphins and seals. The levels of PCBs found in the marine mammals are orders of magnitude greater than the levels found in terrestrial birds and mammals, including humans. In addition, it has been observed that the genetic make-up of marine mammals predisposes them to reproductive failure when exposed to even moderate levels of PCBs.

There are about 1.2 million tonnes of PCBs in the world. Of that total, 31 per cent has been released to the environment (roughly 20 per cent is in the open ocean and 11 per cent in soil and terrestrial sediments. Sixty-five per cent of the worlds PCBs are still in use, or in storage or deposited into landfills. If those PCBs are permitted to leak into the marine environment, then the extinction of marine mammals is inevitable. Although PCB releases into the environment are limited in most western countries, in developing countries such releases (particularly from phased out electrical equipment) are not well controlled. If the released PCBs entered the seas, they would probably prove sufficient to cause the extinction of a wide range of marine mammals, if not all.

The international community must find a way to prevent those PCBs at present stocked on land or deposited in landfills from entering the ocean. In developing countries it is a foregone conclusion that PCBs will escape into the environment unless the cost of preventing this escape is born by an external body. The most appropriate solution is for the PCB manufacturers to "buy back" their products from developing countries. The consequence of failing to control PCB releases to the oceans will be the extinction of marine mammals and the chemical fouling of the ocean fisheries, rendering them unsuitable for use by humans.

PCBs and their Effects

Commercial PCB preparations were first manufactured in 1929. Production peaked between the late 1950s and the early 1970s.

Joseph E. Cummins *is Associate Professor of Genetics at the Department of Plant Sciences, University of Western Ontario, London, Ontario N6A 5B7, Canada*

Raising PCBs:
The Route to the Oceans

PCBs enter the oceans through two main routes: by deposition from the atmosphere and through the drainage of rivers. The best available evidence indicates that about 2 per cent of the PCBs currently entering the oceans do so via rivers, while 98 per cent enter via the atmosphere.

The pervasiveness (and thus the potential impact) of PCBs is clearly brought home by the discovery that rainfall in isolated northern regions of Saskatchewan, Ontario and New Brunswick has been found to contain up to 17 parts per trillion PCBs. The maximum level of PCBs permitted by the Government of Ontario in discharges into the environment is *just one part per trillion*. It is impossible, however, to put an injunction on rainfall.

Recently, it has become clear that even the most isolated regions of the high arctic are being contaminated by PCBs, principally through atmospheric deposition. Biomagnification through the food chain is a particular threat to polar bears, which rely on seal blubber for a major part of their diet. Levels of PCBs in the adipose tissue of polar bears increased fourfold between the years 1969 and 1984. If current PCB inputs continue, the bears will exceed the 50 ppm limit designating them as 'toxic wastes' about the year 2006.

but decreased sharply thereafter upon discovery of the widespread environmental contamination they were causing. PCBs were used in electrical equipment because they were very stable and because they were good insulators. PCBs were also used in hydraulic equipment in factories and in metal finishing. Environmental PCB pollution has been most frequently associated with the manufacture of such electrical equipment as transformers and capacitors, and with automobile manufacture. Most of the PCBs produced are still in use, primarily in older electrical equipment.

Pure PCBs form oils that are heavier than water. They are not very easily dissolved in water but they are easily dissolved in fat or organic liquids. PCBs are very stable in the environment and suffer very little biodegradation. PCBs migrate through the environment via surface waters (normally in association with microscopic soil particles) and via the air.

PCBs are injurious to living beings. They accumulate in fatty tissue and readily pass through the lipid portions of the membranes of cells. It is well documented that PCBs both initiate and promote cancers (in Ontario, PCB-associated cancers of occupational origin are compensated); in addition, they cause birth defects in humans and animals; reduce immune defences and induce hypertension and stroke. Children of mothers who ate fish from the Great Lakes mildly polluted with PCBs (at or below legal standards) have been found to suffer significant learning and behavioural defects. Large human populations in Japan and in Taiwan were exposed to elevated PCBs by ingesting contaminated rice oil. These human exposures clearly established the toxic manifestations of PCBs.

PCBs — a category which includes 209 distinct molecules — can drastically reduce certain bird populations by causing egg shells to grow thin and fragile. They have a hormone-like effect that interfe...

mammals affected by PCBs. They also cause a drastic reduction in fertility in some mammalian males.

PCBs act through the same biological systems as do the more toxic chlorinated dioxins and chlorinated dibenzofurans. Until quite recently it was believed that PCBs were uniformly less toxic than their more potent relatives the dioxins and furans. However, current evidence indicates that most of the toxicity of PCBs resides in a few isomers of the non-ortho chlorine substituted coplanar PCBs, namely 3, 3', 4, 4', tetra (T_4CB), 3, 3', 4, 4', 5-penta (P_5CB) and 3, 3', 4, 4', 5, 5'-hexachlorobiphenyl (H_6CB). These PCB isomers have been found to be toxic to within an order of magnitude of the most toxic dioxin (2, 3, 7, 8-TCDD) and are present at higher levels in human bodies.[1] The most toxic PCB isomers are called "coplanar PCBs" for brevity's sake.

Marine Mammals and PCBs

Until quite recently, PCB accumulation was believed to be greatest nearest the sources of pollution. Recently Tanabe[2] summarised studies showing that cetaceans (including striped dolphins, melon-headed whales and Dalls porpoises) were found to contain higher levels of PCBs than terrestrial mammals and birds *inspite of living in the pristine oceans, far from land-based PCB pollution*. Very high PCB levels were observed in killer whales from the deep ocean — 410 parts per million (ppm) in blubber, for example — and in blue-white dolphins off the coast of Europe (833 ppm). The levels observed far exceed the level (50 ppm) that normally require goods to be labelled and handled in toxic waste containers. Marine mammals may thus, on average, exceed the levels requiring that they be classified as toxic wastes.

In addition to this excessive accumulation of PCBs, marine mammals have a genetically predetermined sensitivity to reproductive impairment by PCBs. The hormone-like action of PCBs causes reproductive impairment in seals (pinnipeds) according to Reijnders,[3] and in porpoises (cetaceans) according to Subramanian *et. al.*[4] According to Tanabe,[5] the induction of reproductive abnormalities by PCBs is related to genetic influences that reduce the capacity for inducing drug metabolizing enzymes when animals ingest phenobarbital and/or methylcolanthrene. Other organisms, such as mink, which are hypersensitive to PCB-induced reproductive failure also have smaller capacities for inducing drug metabolizing enzymes. Interestingly, about one in ten humans of European origin are genetically similar to mink and marine mammals as regards the capacity for inducing drug metabolizing enzymes, while the remainder have much greater capacities. The inference is that one in ten humans of European origin are probably sensitive to PCB-induced reproductive impairment.

In conclusion, marine mammals are accumulating elevated levels of PCBs and the animals are genetically sensitive to PCB-induced reproductive impairment. There is a very real concern that such animals are facing extinction.

Worse to Come?

There are about 1.2 million tonnes of PCBs in the world. According to Tanabe,[6] 65 per cent of that tonnage is either in use in electrical equipment, or stored on land or deposited in

"Marine mammals are genetically sensitive to PCB-induced reproductive impairment. There is a very real concern that such animals are facing extinction."

remainder having been degraded or incinerated.

For the most part, the richer countries have secure control over the 'landlocked' PCBs. The poorer, developing countries (holding about 15 per cent of total world stock of PCBs), however, are unlikely to control the pollutant, and, should the PCBs held by them ever enter the oceans, they would be sufficient to cause the extinction of most marine mammals.

Where PCBs are trapped in sediments and soil, the pollution is localized in 'hot spots' which can be identified and contained. The PCBs in the oceans, by contrast, cannot be contained. Similarly, much of the global PCB burden is now being redistributed by airborne transport. Swackhamer and Hites,[7] for example, observed elevated levels of PCBs in an isolated lake on a large island in Lake Superior which had never experienced extensive anthropogenic activity. In that instance, the PCB transport was airborne and elevated levels were observed in game fish as a result of biomagnification. Indeed, PCBs have penetrated throughout the global environment and now pollute the waters and animals at both poles. However, the northern hemisphere is more polluted than the southern.

The mid-latitudes of the northern hemisphere are far more polluted with PCBs than is the remainder of the world. This distribution is related to the localization of industry. If the further environmental release of PCBs into the environment is prevented, PCB levels would (at least in theory) decline due to dilution and dispersal. However, dilution provides little relief as marine mammals biomagnify PCBs by factors as great as ten million times.

Biomagnification and the surface microlayer

Aquatic contaminants of low water solubility associate with floating particles concentrated at the sea surface. The upper 50 micrometers of water contains pollutants concentrated from atmospheric deposition, terrestrial runoff and sewage disposal. Pollution levels in this surface microlayer exceed those observed at lower levels by orders of magnitude; moreover, water samples from the upper microlayer have induced developmental abnormalities and genetic damage in test animals.[8,9] Pollutants in the surface microlayer tend to pass into the food chain because photosynthetic organisms seek the surface and fish and mammals feed at that layer. Ultimately, in some areas, it may be necessary to 'skim' the oceans' surface to remove dangerous pollutants.

A Scenario for Disaster.

Within the present decade, the majority of electrical equipment containing PCBs will wear out in both industrial and developing countries. If the 15 per cent of world PCBs at present in use, in storage or simply dumped in developing countries were released into the general environment, the extinction of marine mammals would be inevitable. Developing countries, lacking foreign exchange and suffering formidable debt burdens are unlikely to provide funds for environmental protection. Developing countries would undoubtedly be grateful to obtain exchange credits in exchange for their PCBs which could be safely destroyed for the world's benefit. PCB manufacturers should be persuaded to buy back their dangerous products from developing countries. The World Bank should play an important role in ensuring exchange credits for PCBs held by debtors and in convincing the PCBs producers to purchase back their deadly products.

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"Sixty-five per cent of the worlds PCBs are still in use, or in storage or deposited into landfills. If those PCBs are permitted to leak into the marine environment, then the extinction of marine mammals is inevitable."

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OF THE

NATIONAL OCEANIC AND ATMOSPHERIC ADMINISTRATION'S

FINAL REPORT

ON THE

CLINICAL INVESTIGATION OF THE 1987-1988 MASS MORTALITY OF BOTTLENOSE DOLPHINS ALONG THE U.S. CENTRAL AND SOUTH ATLANTIC COAST

MAY 1989

INTRODUCTION

In June 1987, unprecedented numbers of Atlantic bottlenose dolphins, (Tursiops truncatus) began to wash ashore along the coast of New Jersey. In response, Congress mandated the National Oceanic and Atmospheric Administration (NOAA) to investigate the cause or causes leading to what would soon result in the loss of over 50% of the east coast migratory dolphin population.

On February 1, 1989, NOAA announced that the nearly 2 year long investigation into the massive dolphin die-off along the United States east coast during 1987 and 1988 had come to a conclusion. The dolphins had been "poisoned by eating fish tainted by a naturally occurring toxin from 'red tide' algae". According to the press release, "brevetoxin ... killed some of the dolphins directly ... and weakened others making them more susceptible to a host of bacterial and viral infections."

NOAA did not release any supporting data at that time to substantiate their findings. Only after the preliminary report, upon which the press conference was based, had completed a peer review process would a final report be made available to the general public. NOAA estimated that this would take about ten days. The report, Clinical Investigation of the 1987-88 Mass Mortality of Bottlenose Dolphins Along The U.S. Central And South Atlantic Coast (Final Report), was made available nearly two months later.

The purpose of this critique is to analyze the validity of the conclusions of NOAA's Final Report as well as the validity of the investigative directions leading to those conclusions. Although the Final Report does not contain much of the data necessary for a complete review, it clearly bespeaks of an investigation that was biased towards incriminating a natural event or cause to the dolphin mass mortality. As a result, the conclusions of NOAA's Final Report are scientifically unjustified and are not based on a critical analysis of a broad range of possible factors. Indeed, the outstanding feature of the Final Report is its devotion to making a case for brevetoxin as the causative factor while implications of anthropogenic (manmade) chemical contamination were lowly prioritized and largely ignored. In fact, the lack of data presented in the Final Report on chemical contaminants show the investigation, itself, had not attempted to determine the extent to which pollution may have contributed to the dolphins' demise.

The investigations' predispositions are best exemplified by the following points:

a) brevetoxin, found in only 8 of 17 animals, and with limited proof of its chronic ingestion by the dolphins and no proof of its chronic effects was, nonetheless, deemed the proximate cause;

b) organochlorine pollutants, found in high levels in all animals tested and with overwhelming evidence of their toxicity to mammals, were not deemed as being important.

Does this mean that chemical pollutants, such as polychlorinated biphenyls (PCBs), killed the dolphins? Unfortunately, the information uncovered by the investigation and presented in the Final Report does not allow for a definitive answer to be made. But because there is much evidence to show that human-induced environmental stress may have played a role in the dolphin die-off, Greenpeace feels that the investigation into the causes of the mass mortality of bottlenose dolphins is far from over and must be reopened. In the final analysis, the goal has to be the devotion to uncovering or discovering the facts in order to ensure, ultimately, the protection of ourselves and our environment. If human involvement, directly or indirectly, played a role in the dolphin die-off then this must be recognized and addressed.

A. THE IMPLICATION OF BREVETOXIN

1. Events Leading to the Die-off

It has been proposed that a combination of atypical and unique events caused the die-off:

- Ptychodiscus brevis cells (the individual dinoflagellates that produce brevetoxin) from a "red tide" bloom on Florida's west coast were transported by a Gulf current to the east coast during the spring of 1987;

- the cells were consumed by plantivorous fish which were, in turn, either consumed by the dolphins directly, or consumed indirectly through an intermediate vector (i.e. via a predator fish);

- some of the dolphins ingested an acute lethal dose of brevetoxin through consuming contaminated fish while the rest ingested a chronic sub-lethal dose of brevetoxin by consuming contaminated fish over the period of time when both dolphins and fish were migrating northwards during the spring of 1987.

There was no documented east coast bloom during the spring of 1987. However, both the Final Report and statements by the principal investigator, Dr. Joseph Geraci, at the NOAA press conference were not clear when describing what kind of situation was believed to exist off the east coast with respect to the P. brevis cells and brevetoxin. Dr. Geraci did state at that press conference that this particular scenario was caused by "the accumulation of these organisms [P. brevis] at a very opportune time over a longer period of time -- not necessarily a bloom -- but this persistence of these organisms in this precise location where the rest [fish] can pick them up". The Final Report suggests that a east coast bloom existed and remained undetected for 6 months before a filament reached the North Carolina coast in late October of 1987.

Ptychodiscus brevis cells are prevalent in much of the Gulf of Mexico in what might be termed a normal condition of less than 1000 cells/liter. Under ideal water and weather conditions, a "bloom" (more than 5000 cells/liter) can develop sometimes covering areas of thousands of square miles where it can cause massive fish kills. Blooms occur regularly in the Gulf but there have been only 4 recorded cases of blooms, including one in North Carolina in late 1987, on the U.S. east coast since 1970.

Thousands of bottlenose dolphins inhabit the Gulf of Mexico in what appears to be very much the same condition hypothesized as occurring on the east coast during the spring of 1987. Yet there has never been a recorded case of dolphins negatively impacted by brevetoxin-carrying fish. Nor is there any documented evidence of any east coast filter feeding fish, or predator fish, showing brevetoxin contamination in 1987. 4 menhaden, including one in a dolphin, were found with brevetoxin in 1988 and it is theorized that they picked this up from the "red tide" bloom in North Carolina in late 1987.

2. The Analysis of Brevetoxin

In short, the determination of brevetoxin in the dolphins consisted of the following procedure:

- samples underwent 3 purification steps each followed by fish bioassay -- if at any bioassay stage the fish lived (i.e. a negative result), then the test was terminated for that sample;

- only those samples found positive through the third bioassay were subjected to high pressure liquid chromatography (HPLC);

- diagnosis was based on detecting a specific HPLC peak which comigrated with (i.e. agreed with) the brevetoxin standard.

However, much of the process involved with HPLC analysis requires interpretation by an analyst (matching peak retention time with the retention time of the respective standard). The real possibility exists with this type of analysis that several contaminants could coelute simultaneously making it difficult to decipher any given contaminant with a given standard. In addition, the HPLC method for brevetoxins is, itself, plagued with difficulties: lack of reliable reference standard material for this family of related compounds; the stability of the toxins under extraction procedures and; the loss of toxin in preparative steps. These problems make all quantitative values of "brevetoxins" highly doubtful. As well, the Final Report does not specify the components identified in the brevetoxin standards (brevetoxin-B, GB-1, GB-6, etc.), which may vary in distribution and potency. Because of these difficulties, and because of the precedent-setting nature of the report's conclusions, it should have included a description of the HPLC results, including displays of the chromatograms, and an explanation of why the results were interpreted as they were with any caveats on that interpretation.

The results of the brevetoxin analyses, as presented in the Final Report, clearly attest to the difficulties the investigative team faced. 4 of the 17 dolphins that comprised the die-off sample tested positive through the 3 bioassays (i.e. the fish used for the bioassay died) yet the chemical, when analyzed by HPLC, showed no relation to brevetoxin. This could indicate the existence of another contaminant. 3 more that underwent an HPLC analysis after showing positive on all fish bioassays had a peak suggestive of brevetoxin but this peak did not comigrate with the brevetoxin standard. Both these cases suggest the existance of another toxic contaminant in the dolphin liver samples. The Final Report makes no mention of the possible implication of this nor is there any mention of attempts to isolate and identify this compound. 2 more showed positive only on the first bioassay and were concluded to not have brevetoxin but to contain a substance toxic to fish but of unknown consequence to the dolphins. The end result is that only 8 of the 17 dolphins that comprised the die-off sample were found to contain what was deemed to be brevetoxin. Those animals were:

- 2 found near Virginia Beach, from the period of Aug. 8 to 26, 1987;
- 3 found near Virginia Beach, from the period of Sept. 18 to Oct. 8, 1987;
- 3 found along northern Florida, from the period Dec. 13, 1987 to Feb. 19, 1988.

The report pointed out that the positive results found in the three strandings along northern Florida could have come as a result of the brevetoxin bloom in North Carolina during November and December of 1987. However, if this is the case, we have only 5 animals (those from the VA area) that tested positive for brevetoxin before the only recorded bloom on the east coast in 1987.

In terms of the results, it should be mentioned here that 3 of the animals designated as controls actually died during capture near Virginia Beach during the height of the die-off. The Final Report does not give any basis for using these animals as controls rather than as for samples of the die-off. One of those animals tested positive through 3 bioassays but the HPLC analysis showed a peak that did not comigrate with the standard. The 2 others tested negative on the third bioassay. If these animals were included as part of the die-off sample, which appears to be justified, then the die-off sample would comprise 20 animals rather than 17.

From this section, the following assumption would then have to be made in order to follow true to the brevetoxin theory:

a. the apparent delineation of brevetoxin in 8 out of 17 animals (47 %) is enough to conclude that brevetoxin was common to all animals in the die-off.

3. <u>The Effects of Brevetoxin</u>

Direct exposure to brevetoxin for some species of fish (i.e. mullet, catfish) can cause death though the exact mechanism of action is still open to speculation. P. brevis blooms can also cause fish kills by sufficiently depleting the oxygen levels in water. Brevetoxin can accumulate in filter-feeding shellfish which, if ingested by humans, causes nausea, vomiting, diarrhea, numbness and other effects. However, there has not been a recorded case of a human death caused by brevetoxin poisoning. Brevetoxin has been implicated circumstantially with the acute deaths of manatees on the west coast of Florida in 1982 (O'Shea et al., unpublished report, 1983). In addition, "a small number of bottlenose dolphin were reported dead" during a bloom along Florida's west coast in 1947 (Gunter et al., Ecol. Monog. 18, 1948).

Brevetoxin is classified as a neurotoxin (Envir. Health Criteria 37, Aquatic Biotoxins, World Health Organization, 1984) but has not been reported as immunotoxic.

No precedent or studies exist to show what the impact to long-term exposure of sub-lethal levels of brevetoxin would be. The Final Report, however, proposes the following scenario: "A dolphin ... would likely stop eating, eventually exhaust its blubber reserve, and thereby lose its passive buoyancy and thermal shield. The stress associated with these changes alone could set the stage for infection by the ubiquitous opportunistic organisms that were isolated from the affected dolphins" (pg. 18). The Final Report suggests that many of the infections found in the dolphins "may have been related to immunoincompetence" (pg. 15) but that the cause of this could not be established.

Chronic stress can come from a number of factors including harassment, lack of food, short and long-term exposure to environmental contaminants or combinations thereof. Interestingly, there is a wealth of information attesting to the profound immunosuppressive effects of PCBs, of which these dolphins had remarkably high levels. The Final Report, however, chooses to overlook any relationship that may exist between the impact of PCBs and the dolphins' weakened immune system while proposing brevetoxin as the causative agent of immune system suppression.

This brings us back again to the question of bottlenose dolphin populations in the Gulf of Mexico. Here, brevetoxin would be expected to be readily available in the dolphins' food chain (i.e. chronic exposure seems to be a distinct possibility) and yet no die-off as occurred along the east coast has ever been recorded there.

This is hot to say that brevetoxin could not have had an impact on the dolphins during 1987. But any proof showing that brevetoxin has the capacity to negatively impact an animal in this manner does not exist and, as such, any conclusions made have to be regarded as hypothetical.

Curiously, the dolphins appear to have been the only marine species impacted by the 'red tide' bloom and brevetoxin during the spring of 1987. There were no reports of fish-eating animals (eg. seabirds, turtles, other toothed cetaceans) similarly effected.

The distinction between acute and chronic effects of brevetoxin bears further mentioning. The principal investigator, during the press conference announcing the results, stated that "they died, many of them, from direct consequences of the toxin -- others lived long enough to become weakened ..." (press conference transcript, Feb. 1, 1989). Yet, in an apparent contradiction, the Final Report states that "it is clear that most of the dolphin did not die this way" (pg. 17). Whatever the case may be, there is no discussion in the Final Report as to the basis for saying that any of the dolphins died of acute poisoning other than a reference to dolphin KDL 644 that stranded in Florida in early 1988. In this instance, the Final Report queries whether the fact that brevetoxin found in a menhaden inside a dead dolphin that did not have brevetoxin in its liver would be suggestive of acute poisoning. With no explanation, the question is resolved by the next sentence where the report states "most of the dolphins did not die this way" (pg. 17). Interestingly, KDL 644 was found to have an unidentified compound in its liver that killed fish in 3 separate bioassays (see Table 6, NOAA Final Report).

The Final Report states that "death is rapid, and there are no reports of discernable histopathologic changes in acutely poisoned animals" (pg. 17). The Final Report, again referring to dolphin KDL 644 and its suggested acute death, fails to report the necropsy and histopathology results of this animal. Did it have lesions? Was it emaciated? From Appendix 1 of the Final Report it appears that the investigative team were not able to age the animal. Could it have died from old age? Again, whatever the case, for the Final Report to attempt to show that acute death by brevetoxin poisoning occurred in some dolphins by using the example of dolphin KDL 644 suggests a lack of solid evidence.

The Final Report attempts to bolster the brevetoxin hypothesis by citing three cases where circumstantial evidence suggests marine mammal deaths by marine toxins. In all three cases, however, the animal deaths were acute. In the bottlenose dolphin die-off, most, if not all, as described in the Final Report, died of chronic conditions.

The Final report, in attempting to support the brevetoxin theory, made the following assumptions:

a. there were acute deaths by brevetoxin poisoning;

b. the acute and chronic conditions noted in the dolphin can be traced to direct and indirect actions of brevetoxin;

c. chronic exposure to certain levels of brevetoxin can weaken dolphins in such a way as to allow for the invasion of pathogens.

B. THE IMPLICATION OF MANMADE CONTAMINANTS

A bias away from implicating manmade contaminants is strongly evidenced in the Final Report. This is important to note because if brevetoxin did play a role in the dolphins mass mortality, its effects may have been intensified by other toxic compounds. The outstanding feature of the data presented in the Final Report is the high levels of PCBs found in the dolphins including one with 6800 ppm in the blubber (the highest concentration of PCBs ever found in a marine mammal. Other dolphins analyzed showed levels which ranged from 13 to 620 ppm in the blubber.

1. The Analysis of Contaminants

The Final Report lacks data and discussion on the analysis of contaminants as a whole. The principal investigator stated that over 40,000 chemical compounds were analyzed for (Marine Mammal Commission briefing, Sept. 10, 1987) yet no mention is made of this in the Final Report. The lack of data and discussion makes it impossible to assess the rationale for excluding the impact of contaminants other than the 9 that were mentioned. The Final Report should have laid out the types of analyses done for all those compounds along with a discussion as to the accuracy and meaningful conclusions, if any, that could be drawn from those results. Rather, the report produces data and discussion that supports a brevetoxin hypothesis only.

Whether the report mentions it or not, the meaningful analysis for 40,000 + potential contaminants is a difficult, if not impossible, task. Even the most sophisticated analytical equipment and comprehensive data base cannot quantify many of the compounds that may appear from an analysis. As well, analytical procedures for many compounds have to be tailored accordingly in order to provide accurate results (Swallow et al., Environ. Sci. Technol. 22; 1988).

The omission of any reference to other chemicals is all the more surprising when the investigative team itself acknowledges that what they were looking for was a point source contaminant or poison. To this effect, there was no discussion as to the possibility of caustic contaminants directly impacting the dolphin's skin. Direct contact with such a chemical may have resulted in skin aggravation which in turn produced lesions that permitted the entry of opportunistic bacteria. This, as one of many possible scenarios, would have brevetoxin playing a subordinate or perhaps even no role in the dolphin mass mortality.

The analysis of contaminants, or lack of, has other implications in determining the role of manmade chemicals in the die-off. Some genotoxic compounds (chemicals impacting genetic material) such as benzo(a)pyrene (BaP) and the related polycyclic aromatic hydrocarbons (PAHs), produce immune toxicity. Yet as these are readily metabolized by mammals, they are extremely difficult to determine with conventional techniques. Low or undetectable levels may exist in organs or blubber yet there may have been corresponding and extensive gene damage that would only be noticeable through a specific analysis to determine that compound's binding to DNA. Indeed, through such a technique was it determined that at least some St. Lawrence beluga whales had significantly damaged DNA through contact with B(a)P (Martineau et al., J. Comp. Path.98: 287-311, 1988). The manmade PAHs are ubiquitous in many marine environments including areas along the U.S. east coast (NOAA Technical Memorandum NOS OMA 44; 1988).

Through this type of study it would have been possible to have better indicators from which to assess the overall health of the dolphin population before the die-off. This is important because a possible scenario for the die-off may well be that brevetoxin played a role only because the population as a whole was in a compromised state of health. This scenario is virtually excluded from the Final Report (see Section B 3, this paper).

Marine mammals are especially vulnerable to the effects of pollution because of their habitat and their nutrition. PCBs, DDT, the dioxins, the hexachlorobenzenes, dibenzofurans and some of the products derived from them are all important, persistent, highly toxic compounds now found in the environment. Because they do not readily break down, they move up food chains to the top predators such as seals, sea lions and dolphins. Many of these contaminants are lipophilic ("fat-loving") and therefore will accumulate in the ample fatty tissues of these marine mammals. The evidence showing the danger that many of these compounds pose to the overall health of a given animal is almost overwhelming. To that extent, it is surprising that only the analytical results of three organochlorine compounds were presented in the Final Report.

2. Brevetoxin vs. Manmade Contaminants: An Evolutionary Perspective

The Final Report's focus on the impact of sublethal chronic doses of brevetoxin at the expense of sub-lethal chronic doses of organochlorines (organochlorines are specifically mentioned here because high levels were noted in the Final Report) is not justified from an evolutionary point of view. Dolphins may well have evolved in conjunction with brevetoxin for millions of years whereas PCBs, for example, have been in the environment for little more than 50. An example of evolutionary efficiency with regards to detoxification can be seen with marine mammals and their contact with mercury which is naturally found in high levels in many marine environments. Mercury by itself, either from natural or manmade sources, is not considered highly toxic. It is, however, readily transformed by microorganisms into highly toxic methylmercury where it becomes concentrated in fish and, subsequently, in marine mammals. Yet marine mammals can demethylate mercury very efficiently before it is distributed throughout the rest of their bodies. The same amounts of methylmercury in terrestrial mammals would have a serious impact.

In this regard, the Final Report's attempt to relate the effects of levels of brevetoxin known to cause illness in man with levels found in the dolphins is not completely justified. This is not to state that brevetoxin cannot have an impact on marine mammals or, in this case, could not have played a role in the dolphin die-off. But, from an evolutionary perspective, it would seem likely that the dolphins would be able to more readily detoxify brevetoxin or that they would be relatively more tolerant to it in their food than man and other terrestrial mammals. Whatever the case, the toxicity of brevetoxin to dolphins is totally unknown.

3. The Implication of Organochlorine Compounds

As mentioned earlier, the levels of organochlorine compounds (PCBs, DDE and chlordane) found in the dolphins are among the highest ever found in a marine mammal population. However, comparisons with concentrations from other similar animals are very difficult to make. It is apparent that some or most of the dolphins involved in the die-off suffered prolonged illness during which they undoubtedly mobilized their blubber stores. This would probably have had two effects: first, as the blubber volume diminishes, the concentration of the organochlorine compounds within it will increase; and second, the compounds are released into the bloodstream and are accumulated in the liver. Because of this, it is not possible to determine what the original concentrations were before the die-off began. Nonetheless, the observed concentrations of organochlorine compounds are very high.

The Final Report, in addressing the levels of PCBs, states that they "can be harmful following both acute and chronic exposure" (pg. 16) and that "typically affected are liver and skin, and nervous, reproductive, and immune systems" (pg. 16). However, further discussion on PCBs and the other organochlorines and their possible role in the die-off is very brief and was centered only on their effects upon mobilization from the blubber when the animals were sick. Here, the Final Report says that toxic compounds may have been released "into vital, perhaps more critical organs such as liver" (pg. 16) and that "it is conceivable that blood levels [of PCBs] rose and were sustained long enough to exert an effect" (pg.16).

The Final Report makes no mention whatsoever of the possible impact of organochlorine compounds on the dolphins (e.g. such as on their immune or hepatic systems) before the die-off even though overwhelming evidence exists attesting to the severe impact of sub-lethal chronic exposure to these compounds (see Appendix I). Could the dolphins, for example, have been weakened by chronic exposure to PCBs making them susceptible to an environmental disturbance (e.g. brevetoxin) that normally would not have negatively affected them? This would even be true of the calves which receive organochlorines through the fat-rich milk provided by their mothers. Or could the organochlorines have caused liver dysfunction after the animals had been stressed and when blubber stores, including the organochlorine compounds, were mobilized? In this scenario, liver dysfunction could have impaired or limited detoxification of not only brevetoxin but also bacterial endotoxins (the poisonous substances produced by the microbes found to be so prevalent in the diseased animals) and the organochlorines themselves. The dolphins may, therefore, have been affected by the high levels of contamination they possessed before, as well as during, the die-off. And again, these contaminants may have predisposed them to other factors which finally, in conjunction with the contaminants, precipitated the mass mortality.

Another instance where the Final Report has apparently incorrectly assessed the possible action of organochlorine compounds within a system is found when it states that the presence of high levels of organochlorine compounds in an animal's blubber "may not pose a risk" (pg. 16) to the animal under stable conditions. There is much evidence to show that this is not the case and that blood serum levels of halogenated biphenyls are related to levels in adipose tissue (Kreiss et al.; Arch. Envir. Health 37:141, 1982). Another study (Reijnders, Neth. J. Sea Res., 1980) states that "the adipose tissue is not an inert depot locking up chlorinated hydrocarbons beyond the period of pregnancy and lactation".

Even though the Final Report alludes to a possible role of contaminants in the die-off, the NOAA press conference does not. In fact, it is made very clear how NOAA views the role of contaminants in the mass mortality:

Question (unnamed journalist): "Could we say that pollution had nothing to do with the die-off?"

Answer (Dr. Joseph Geraci): "Yes. These dolphins died of brevetoxin intoxication and poisoning..."

(NOAA press conference transcripts, Feb. 1/89)

The lack of discussion in the Final Report about, in this case, PCBs, is clearly not justified considering the body of information that exists showing the negative impact of chronic sub-lethal ingestion of these compounds on mammals.

4. <u>Organochlorine Compounds found in Captive Control Animals</u>

3 captive bottlenose dolphins were used as controls for the analysis of chlorinated hydrocarbon residues. These residues found in the liver were very high ranging between 34 and 222 ppm lipid weight. The average level of, for example, PCBs in the captives was 109.2 ppm lipid weight which was higher than the average for mature females and almost identical to immature females in the die-off. The Final Report postulates that the high liver levels in the die-off victims came about as the animals mobilized lipids during stress brought on by ingestion and contact with brevetoxin. But this does not explain why there were high levels in the captive control dolphins whom, according to the Final Report, would have had "no need to mobillize blubber fat which would deliver the compounds to liver for excretion" (pg. 16).

The Final Report fails to give any other information on those 3 control dolphins. Were these animals autopsied? If not, then this would be a major flaw in the methodology because it would be impossible to compare lesions between the controls and the die-off victims. After all, if the two groups had similar lesions then a case for brevetoxin would be even further weakened. If the controls were autopsied then why weren't the results mentioned in the Final Report? Failure to mention lesions in the captive animals when both they and the die-off victims were found with the same contaminants would be a major omission of data.

There are further questions regarding the controls. How did they die? How healthy were they before they died? How old and what sex where they? Where did they come from? These questions are all important if one wants to determine the possible role of at least some contaminants in the die-off.

5. <u>Relating Known Effects of Compounds between Species</u>

The Final Report, in attempting to nullify the impact that PCBs may have had in the die-off, states that "we cannot categorically relate any of the conditions observed in the dolphins to the known effects of these compounds because of vast differences in response within and between species" (pg. 16). This may be true 'categorically' but the Final Report fails to mention that PCBs have been shown to produce atrophy of lymphoid tissue (the tissue responsible for immune function) in mammal species where this response was measured in conjunction with PCB exposure. Only the amount of PCBs required to produce immune dysfunction varies with the species.

While comparing the known effects of PCBs between species is not feasible according to the Final Report, comparing the effects of brevetoxin between species apparently is. The report devotes a paragraph (pg. 19) relating dosage with response in mice and humans to possible impact on the dolphin.

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6. Mobilization of Compounds from Blubber to Liver

The Final Report states brevetoxin "may be stored in fatty depots and mobilized along with fats as the animal draws on these reserves" (pg. 19). This would explain the discovery of brevetoxin in nursing calves who would receive it through the mother's milk. This, however, is exactly what occurs with other lipid soluble contaminants such as PCBs. This begs the question of whether or not brevetoxin was present in the liver only because something else had stressed the animals thereby releasing brevetoxin that had been ingested sometime earlier. A possible scenario (out of many) for the die-off could then be that some stress caused the dolphins to mobilize their fat which then delivered PCBs, DDT, chlordane and brevetoxin to critical organs. This sufficiently weakened the animals allowing for attack from opportunistic pathogens.

CONCLUSION

The outstanding feature of the Final Report into the Investigation of the 1987-88 Mass Mortality of Bottlenose Dolphins along the U.S. Central and South Atlantic Coast is its bias towards making a case for brevetoxin as the causative factor of that mortality. This bias has resulted in the brevetoxin theory presented as virtually a fact without the evidence to draw such a conclusion. Furthermore, this was done at the expense of a balanced and wide ranging discussion into other equally possible scenarios or contributing factors.

Though the Final Report arrives at the conclusion that brevetoxin was probably the causative agent, that conclusion itself is incumbent on a number of assumptions:

1) that the apparent discovery of brevetoxin in 8 out of 17 animals is enough to conclude that brevetoxin was common to all animals in the die-off;

2) that the chronic exposure of the dolphins to brevetoxin via their food can produce the conditions that would make them susceptible to the invasion of opportunistic bacteria; and subsequently,

3) that the levels of brevetoxin found in 8 dolphins are of a high enough order to assume that those levels would correlate with chronic stress and possible reduced immune system efficiency;

4) that the acute and chronic conditions noted in the dolphins can be linked to direct and indirect actions of brevetoxin; and

5) that there is enough evidence for brevetoxin to conclude that no other factor(s) triggered the die-off.

The Final Report states that "the dolphins apparently were poisoned by brevetoxin" (pg. 1) and that there is evidence implicating brevetoxin as the "proximate cause" (pg. 1). However, there has been no documented scientific research into the possible effects of sub-lethal chronic doses of brevetoxin on mammals. The Final Report compounded this by assuming that the levels found were representative of a significant toxic load and that this load was common to all animals even though brevetoxin itself was found in only 8 of 17. Finally, the report held that there were no other primary factors in the die-off.

In making a case for brevetoxin, the Final Report omits a critical discussion of the possible role of the record levels of some manmade compounds found in the dolphins. For example, PCBs have definitively been shown to be immunotoxic and hepatotoxic: the immune and detoxifying systems of many of the die-off dolphins were shown to be severely compromised. The Final Report presented no data on the toxicological analyses as a whole nor did it show any evidence that specific analyses were done for many compounds such as: dioxins, including its most dangerous form 2,3,7,8 tetrachlorodibenzo-p-dioxin; the polycyclic aromatic hydrocarbons; and the polychlorinated dibenzofurans. This is representative of a larger problem within the investigation: human-induced environmental stress was presupposed to not have played a role in the die-off. As a result, the rigorous scientific investigation that would have been necessary to uncover possible roles of man-induced environmental stress never occurred.

The Final Report does not address a glaring question precipitated by the brevetoxin theory: why has there not been a documented case of a similar bottlenose dolphin die-off in the Gulf of Mexico where P. brevis is indigenous and is continually available for accumulation into their food chain?

The Final Report makes no attempt to discuss or raise other scenarios into the causes of the mass mortality. On the basis of the information presented and on the theories proposed, there is little on which to assert that brevetoxin was responsible. It rests that brevetoxin may have been a factor in the die-off but it remains equally possible that brevetoxin played a secondary role or that it had no role at all.

Incredibly, NOAA chose not to publicly release the levels of contaminants found in the dolphins even though the pattern of high contamination was known by late 1987. Was this information withheld for fear of alarming the citizens of the east coast? To that effect, the contaminant levels found in the dolphins are representative of an east coast ecosystem that is under toxic attack. The die-off, and other recent marine anomolies, points to a possible unravelling of this system's integrity. We feel there is great cause and need for alarm.

In summary, the investigation into the causes of the die-off was unbalanced and thus inadequate. It can be considered only as a first step towards determining those factors that contributed to or caused the east coast dolphin mass mortality of 1987/88. As it stands, the Final Report of the investigation raises far more questions than it answers and it shows an investigation strongly predisposed to implicating natural phenomena while dismissing the potential role of man-induced causes. It is hoped that that the cloak of secrecy that surrounded this investigation will be lifted to allow for a more open exchange of data, findings and interpretations among a broader spectrum of participants.

We submit there is an urgent need for the investigation to be re-opened. The status of the remaining east coast dolphin population is critical and dolphins continue to wash ashore with symptoms that depict longterm chronic illness. The remnant population remains highly susceptible to additional perturbations. The longterm future of the east coast dolphin population and, indeed, the east coast ecosystem is at stake.

APPENDIX I

PCBs have been related to a number of dysfunctions in fish and aquatic organisms, birds, and mammals, including man. Following is a brief list of some effects on mammals arising from chronic exposures to sublethal levels of PCBs:

Animal	Effects	Reference
monkey	immunosuppression	Truelove et al.,1982
mouse	malformed fetus	Marks et al.,1981
mouse	enlarged liver	Biocca et al.,1981
mink	reduced reproduction, kit deaths	Horshaw et al.,1983
mink	hepatic necrosis	Platonow et al.,1973
mink	reproductive dysfunction	Jensen et al.,1977
pig	gastric erosions, septicemia	Hansen et al.,1975
guinea pig	immunosuppression	Vos et al.,1981
rat	immunosuppression	Van Velson et al.,1984
rabbit	damaged liver	Koller et al.,1973
man	liver disorder, chloracne	Meigs et al.,1954
man	liver enzyme induction	Fischbein et al.,1979
man	altered liver biochemistry	Chase et al.,1982

Clearly, results related from laboratory testing for PCB effects on laboratory animals, or from health studies on man, to a possible impact on the dolphin has its limitations. As well, results from laboratory testing for PCB effects alone are usually not entirely appropriate in the field where a multitude of chemicals can act upon an organism -- either additively, synergistically or antagonistically. Still, there is overwhelming evidence to show that long-term chronic exposure to sublethal levels of organochlorines, or in this case, PCBs, has a severe impact on many life forms.

APPENDIX II

POLLUTION'S EFFECTS ON MARINE MAMMALS

From the evidence gathered from other species and the contamination levels reported from marine mammals around the world there is clearly cause for concern. Indeed, there is considerable evidence that various populations are being directly impacted by manmade pollution.

Dutch Common Seals

Of two groups of Dutch common seals, one fed on fish from the polluted waters of the Wadden Sea (where the seals live) and the other on fish from the comparatively cleaner northeast Atlantic, the former showed significantly lower reproductive success. PCB's are thought to be responsible for the low rate of reproduction in Dutch common seals (Reijnders; Nature 324; 1986).

Baltic Ringed Seals

In the seriously polluted Baltic sea, ringed seals' reproductive success is low. In 1976 only half the females of reproductive age were pregnant and half of the non-pregnant females had enlarged and scarred wombs indicating that embryos had been reabsorped or aborted. Animals exhibiting these features had significantly higher tissue concentrations of DDT and PCBs than normal pregnant females (Bergman; I.C.E.S., C.M.; N:10; 1981). These pathological changes were also found in grey seals from the Baltic area and harbour seals from the Swedish West Coast. The Scandinavian scientists responsible for this study concluded that it was "strongly indicated" that PCBs were responsible for the seals' reproductive failure (Helle et al.; Ambio 5: 261-263; 1976).

California Sea Lions

Similarly, in the 1970s, many sea lions off California produced premature young. In those females which successfully gave birth, PCB and DDE levels were 6.6 and 8 times lower than those which failed. Many animals were infected with Leptospira bacteria, a pathogen known to interrupt pregnancy. It has been suggested that the immunosupressory effects of organochlorines might be facilitating infection and then premature pupping (Delong et al.; Science 181; 1973).

More recently, in 1988, two separate diseases seem to have been affecting them. Between June and November, 100 sea lions suffering once more from Leptospira infection were taken into the Marine Mammal Center outside San Francisco. A researcher from the University of California believes that the disease only has significant impact when the immunity of the sea lions drops sufficiently. The second illness seen in 1988, which causes seizures in sea lions and fur seals, seems limited to an area of highly industrialized coast. The symptoms appear very similar to those of heavy metal poisoning.

North Pacific Dall's Porpoises

Pollution-induced interference with sex hormones is also reported in porpoises. In fact, it has been suggested that the small whales are even more vulnerable to persistent organochlorine contamination than seals because their enzyme systems are less capable of destroying them. Japanese scientists, in 1987, showed that tissues of Dall's Porpoises from the northwestern North Pacific had high organochlorine concentrations which seemed to correlate with low levels of the male sex hormone, testosterone (Subramanian et al.; Mar. Pollut. Bull. 18: 643-646; 1987). This strongly suggests that existing levels of some organochlorines can cause imbalances in sex hormones resulting in reproductive abnormalities in the wild.

St. Lawrence Beluga Whales

In whales and dolphins, probably the best documented case of the detrimental effects of pollutants is that of the beluga whales in the St. Lawrence River, Canada. The population is close to extinction. Analysis of the tissues of stranded beluga (of which there have been over 90 in the last seven years) has revealed the presence of high levels of organochlorine compounds. Scientists studying the population state that "organochlorine contamination should be considered as a prime cause for the low recruitment observed in this population" (Martineau et al.; Arch. Environ. Contam. Toxicol. 16; 1987). Concentrations of organochlorines are comparable to those reported from the east coast bottlenose dolphins involved in the die-off.

The beluga have been found to be suffering from a wide range of acute and chronic diseases including hepatitis, dermatitis, septicaemia, perforated gastric ulcers, pulmonary abscesses, and bronchial pneumonia. One beluga was found with bladder cancer which was postulated to be a result of polycyclic aromatic hydrocarbon (PAH) and organochlorine pollution (Masse et al.; Arch. Environ. Contam. Toxicol. 15; 1986). In addition, scientists studying this population state that the occurrence of PAH - metabollites and high concentrations of organochlorines in these animals "suggest an important role of industrial contaminants in the recent decrease of this population" (Martineau et al.; J. Comp. Path.98; 1988).

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NATIONAL OCEANIC AND ATMOSPHERIC ADMINISTRATION

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"Red Tide" Caused Dolphin Deaths in 1987

RELEASE: 11:30 a.m., Wednesday, February 1, 1989

Hundreds of bottlenose dolphins that died off the east coast of the United States during the summer of 1987 and into early 1988 were poisoned by eating fish tainted by a naturally occurring toxin from "red tide" algae, according to the National Oceanic and Atmospheric Administration (NOAA).

The red tide alga, known as Ptychodiscus brevis, produces the powerful poison, brevetoxin, which killed some of the dolphins directly, NOAA said, and weakened others making them more susceptible to a host of bacterial and viral infections.

NOAA reported that this is the first known instance of the toxin's being transmitted to a mammal through tainted fish.

The toxin itself was confined to the liver and other viscera of the fish. It is not present in the flesh and poses no threat to humans eating fish fillets, NOAA said.

The toxin was carried up the coast by fish -- possibly menhaden or Spanish mackerel that had eaten menhaden -- that had consumed the algae.

Red tides are normally confined to the Gulf of Mexico, although occasionally such algal blooms can be carried around Florida and swept north along the Atlantic coast by the Gulf Stream.

Dead dolphins first began washing ashore in southern New Jersey in late June 1987. In early August, NOAA and the Marine Mammal Commission assembled an investigative team in Virginia Beach, Va., to examine stranded dolphins, collect tissue samples and begin an analysis that would eventually involve almost 350 dolphins in thousands of separate tests.

(more)

The team was headed by Dr. Joseph Geraci, a veterinarian working at the University of Guelph in Ontario, and at one point involved more than 100 volunteer scientists and others at dozens of federal, university and private agencies and laboratories.

The brevetoxin analyses were carried out in the laboratories of Dr. Dan Baden at the University of Miami.

By March of 1988, when the event ended, about 740 dolphins had washed ashore from New Jersey to Florida. NOAA estimates a substantially larger number died and were lost at sea.

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TESTIMONY
OF
WILLIAM E. EVANS
UNDER SECRETARY FOR OCEANS AND ATMOSPHERE
NATIONAL OCEANIC AND ATMOSPHERIC ADMINISTRATION
U.S. DEPARTMENT OF COMMERCE

BEFORE THE
SUBCOMMITTEE ON OVERSIGHT AND INVESTIGATIONS
COMMITTEE ON MERCHANT MARINE AND FISHERIES

U.S. HOUSE OF REPRESENTATIVES

MAY 9, 1989

Mr. Chairman and Members of the Subcommittee:

Thank you for the opportunity to be here today to review our understanding of the events surrounding the deaths and stranding of Atlantic bottlenose dolphins on the east coast during 1987 and 1988. This was a unique event, which led us all to wonder about what in the ocean ecosystem in which these animals live could possibly have caused these startling happenings.

I would like to spend a few minutes this afternoon reviewing the events as they happened and how we responded, and comment on how we view the situation today.

A marine mammal stranding is not a unique event -- they happen almost routinely along coastal waters. Various groups, including the Smithsonian Institution, monitor these and respond as best they can.

During the second week of July 1987, the Smithsonian Institution called us to report that for some reason large numbers of Atlantic bottlenose dolphins were stranding in the lower Chesapeake Bay and Virginia Beach areas. At the same time, the New Jersey Marine Mammal Center, a private organization, began reporting a significant increase in dead Atlantic bottlenose dolphins along the Atlantic coast from Delaware Bay to New Jersey.

We began monitoring the situation closely, as did the Marine Mammal Commission. During the second week of August, the Commission convened a special clinical investigation team, which included the Smithsonian Institution, the Environmental Protection Agency, and the Animal and Plant Health Inspection Service of the Department of Agriculture. The clinical investigations were considered necessary because in both the Virginia Beach and New Jersey areas, the large number of animals washing ashore were obviously seriously ill, and dying as they came onto the beach. The leader selected for the team was Dr. Joseph Geraci of the University of Guelph, a well-known marine mammal veterinarian. The largest numbers of stranding were occurring in Virginia Beach, and so the team began its necropsy work in laboratory facilities provided by the U.S. Navy's Little Creek Amphibious Base.

What we saw happen was an unprecedented die-off of Atlantic bottlenose dolphins that began in the Delmarva area the early summer of 1987 and progressed on in time through the summer, fall and winter, gradually moving southward. It became obvious to us as this was developing that we would have to spend some effort analyzing the experiences of the Response Team and the tremendous amount of data that came out of their activities. Along with the Marine Mammal Commission and the Office of Naval Research, NOAA developed a plan for funding this follow-up work. Because of his reputation, his familiarity with the events, and the quality of the leadership he showed on the Response Team, we contracted with Dr. Geraci to oversee the follow-up studies and prepare a report on these events, and what might have caused them.

We received Dr. Geraci's final report two weeks ago. His report, which he will discuss with you, summarizes the events from the early summer of 1987 to the early spring of 1988, outlines the methodology for conducting the studies, details the findings of the studies, and discusses their implications.

The report concludes that there is evidence that the dolphin mortalities may have been caused by brevetoxin -- a neurotoxin arising from red tide -- moving up the food chain. Brevetoxin also acts directly upon the respiratory system and has been linked to fish die-offs and respiratory dysfunction in swimmers. To say the least, this hypothesis has caused quite a controversy, even before the report was released. The draft report was circulated to peer scientists for review and comment. Dr. Geraci has considered those comments, yet, as he will point out, he remains convinced of the validity of his observations and conclusions.

We in NOAA accept the report for what it is -- the best judgment of our consultant, interpreting available data, and looking for a conclusion that best fits the available information. Because of our respect for Dr. Geraci and the Response Team, we are confident that the analyses were done competently. The conclusions of the report present us with a challenge to investigate new possibilities in cases of marine mammal stranding, and particularly those involving Atlantic bottlenose dolphins.

Brevetoxin poisoning, frankly, was not considered to be a cause of marine mammal stranding and deaths in the past. It is not something we have looked for in these cases, and it may have been a cause that we simply missed in previous stranding.

I would also note that the Report does not rule out other contributing factors, and we need to be aware of these as well as we plan our future research and monitoring activities. Many have wondered why ocean pollution was not treated more significantly as a potential cause. I will let Dr. Geraci comment in detail, but let me also say that I have great respect for his judgment that while we cannot rule out the contributing influence of the high contaminant levels, we cannot, based on available data, tie them to these dolphin mortalities.

Let me reemphasize NOAA's concern for the health of the marine environment. The report recognizes the need to investigate the dynamics of this epidemic, and further, I would certainly support this need.

Mr. Chairman, this was a unique event, both perplexing and alarming. The Response Team has given us a detailed report of their investigations and findings, including a conclusion as to how this event may have happened.

This concludes my prepared statement, Mr. Chairman. I will be happy to answer any questions you or the Members of the Subcommittee may have.

TESTIMONY

TO: THE OVERSIGHT AND INVESTIGATIONS SUBCOMMITTEE
COMMITTEE ON MERCHANT MARINE AND FISHERIES
U.S. HOUSE OF REPRESENTATIVES

Re: CLINICAL INVESTIGATION OF THE 1987-88
MASS MORTALITY OF BOTTLENOSE DOLPHINS
ALONG THE U.S. CENTRAL AND SOUTH ATLANTIC COAST,
FINAL REPORT

From: Dr. Theodore J. Smayda
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Kingston, Rhode Island 02881

Date: 9 May 1989

The reported die-off of over 740 bottlenose dolphin, Tursiops truncatus, along the Atlantic coast from New Jersey to Florida from early June, 1987 until March, 1988 is unprecedented. The inshore stock of this coastal, seasonally migratory species numbered about 3,000 to 5,000 animals (Kenney, 1989). Thus, this die-off represents a significant population loss of this stock, estimated by some to be upwards to 50% of the total population (Scott et al., 1988). Equally stunning is the provocative conclusion that a biotoxin was the proximate cause of this die-off, namely brevetoxin, a neurotoxin produced by the photosynthetic, planktonic microalga Ptychodiscus brevis. This microalga is a member of the phytoplankton, the basis of all foodwebs in the sea. Paradoxically, some members of the phytoplankton produce compounds that are among the most potent natural toxins known. Under certain conditions, and for as yet obscure reasons, such toxic species proliferate wildly causing seawater discoloration -- "red tides". Such toxic blooms can extend over thousands of square kilometers and persist for prolonged periods. Transfer of "red-tide" toxins through the foodweb leading to finfish die-offs have previously been documented, such as massive herring kills in the Bay of Fundy. Autopsies of 14 humpback whales and two minke whales, which died during the late fall of 1987 off Cape Cod, revealed the presence of "red-tide" toxins. These whales were feeding on mackerel, a vector of "red-tide" toxin transfer, suggesting that their deaths were attributable to a phytoplankton biotoxin. Thus, there is some evidence that finfish die-offs certainly, and possibly marine mammal deaths result from foodweb transfer of toxins produced during certain phytoplankton blooms.

It is my conclusion that the evidence provided by the Gerasi report attributing the proximate cause of the 1987-88 dolphin die-off to foodweb transfer of the "red-tide" toxin, brevetoxin is, at best, tenuous rather than conclusive or convincing. I base this conclusion from the vantagepoint of my professional experience with "red-tide" population dynamics and foodweb transfer, rather than as a toxicologist, pathologist or veterinarian, areas in which I have no expertise.

Several problems are associated with the attribution of brevetoxin as the most probable cause of the mortality, namely the source and delivery mechanisms of this toxin. Ptychodiscus brevis, the source of brevetoxin, is found principally in the Gulf of Mexico. In October 1987, an extraordinary, previously unrecorded, anomalous bloom of this species occurred near Cape Hatteras, the most northerly penetration of this species along the Atlantic coast recorded to date. There are no known producers of brevetoxin in the indigenous phytoplankton along the U.S. Atlantic coast. It is notable that about 180 dolphin deaths were recorded along the New Jersey - Virginia coastline between May - September; that is, well in advance of the mid-October Cape Hatteras Ptychodiscus brevis bloom. Since dolphins can not ingest Ptychodiscus brevis directly, its toxin must be ingested with its prey. (The actual diet of the bottlenose dolphin is poorly known (Kenney, 1989). Menhaden, which are planktivorous and potential toxic vectors, have been implicated; indeed, the occurrence of brevetoxin was reported in one specimen. However, menhaden migrate northward during the spring, with little or no movement north or south of Cape Hatteras from about June to November (Nicholson, 1978). A migration of toxin-containing menhaden into the northern dolphin die-off region was, therefore, unlikely. Moreover, the northward migrations of bottlenose dolphin is a spring event (Kenney, 1989), thus recruitment of poisoned dolphin during this period, followed by their localized death is also problematic. The problem, therefore, is that not only are there no brevetoxin-producing phytoplankton species over much of the die-off range, but that the migration patterns of both dolphin and implicated prey-species, such as menhaden, are not consistent with the notion that the foodweb transfer of brevetoxin was the cause of the dolphin die-off. The occurrence of brevetoxin in eight dolphins is not challenged. Adventitious, occasional toxin accumulation would not be surprising. However, neither appropriate toxin sources and foodweb transfer of the required magnitude, nor continuous toxin delivery from ingested carrier-fish over the > 2000 km die-off distributional range has been shown with the data at hand, nor can be developed without serious disregard of current knowledge regarding toxic dinoflagellate blooms. I remain skeptical, therefore, that brevetoxin was the responsible, lethal factor. Other reasons can also be mustered.

There is a related matter of extraordinary consequences occurring in parallel with catastrophic marine biotic events (such as the bottlenose dolphin die-off), also relative to nuisance phytoplankton blooms in the sea, which I wish to bring to your attention.

An epidemic of nuisance phytoplankton blooms is spreading throughout the sea accompanied by anoxia; marine mammal, fish and invertebrate die-offs; human deaths and illness, and trophic dysfunction. Regions previously free from toxic phytoplankton blooms now suffer such blooms; species previously benign have become toxic or nuisances; in many regions the frequency and intensity of red-tide outbreaks have been increasing; human deaths due to paralytic shellfish poisoning are increasing. Bloom events are normal aspects of phytoplankton dynamics essential to marine foodwebs, but blooms collectively known as "red-tides" represent population explosions of species which are undesireable or toxic to grazers.

A significant global increase in kills of commercially important finfish and shellfish, both natural and cultivated stocks, has accompanied the global surge and spreading of nuisance phytoplankton blooms. Remarkable die-offs of whales and dolphins have recently been linked to toxic phytoplankton blooms for the first time. Enormous financial losses have resulted to commercial fisheries and associated industries, sometimes exceeding $100 million per bloom outbreak. Marine aquaculture is presently an uninsurable activity because of the highly unpredictable, episodic nature of lethal "red-tide" blooms. Curiously, finfish and shellfish aquaculture activities themselves frequently stimulate "red-tide" outbreaks in the growth area!

Red-tide outbreaks are not a new phenomenon; historical references to such blooms date back to Homer's Iliad and Odyssey. Episodic red-tide blooms are natural events. What is new is their global spreading, increased frequency and associated catastrophic die-offs of marine animals. Red-tide outbreaks are not restricted to dinoflagellates. Brown, yellow, white and green water discolorations accompany bloom events of other phytoplankton groups. What is new is that groups previously considered to be benign now produce inimical blooms. Diatom blooms, for example, have caused fish-kills and mussel toxicity leading to human death, amnesia and epilepsy. Red-tide blooms historically have been primarily colder water phenomena. What is new is their present proliferation in Tropical and Sub-tropical waters accompanied by increased outbreaks in Temperate and Boreal seas. The eastern coastal waters of the U.S. historically had been relatively free of toxic red-tide outbreaks. What is new is that since 1972 there have been at least six major toxic blooms in the waters stretching from Massachusetts to North Carolina.

In September 1972, New England had its first serious paralytic shellfish poisoning epidemic following a red-tide. At least 26 people were poisoned, and the clam-beds were closed down at a revenue loss of about $1 million per week. The causative organism, Gonyaulax tamarensis var. excavata has since spread, causing recurrent toxic blooms along much of the New England coast, causing periodic closure of the shellfish areas.

During the summer of 1976 a large, anomalous bloom of the dinoflagellate Ceratium tripos occurred in the New York Bight. Ungrazed, its growth eventually became limited by nutrients, such as nitrogen, the population sank into bottom waters, rotted, used up the available oxygen and caused anoxia. Significant mortality of commercially important fishery species, such as surf clams, scallops, lobster and certain finfish ensued. The estimated commercial revenue loss was $67 million. I have been told that environmental conditions similar to 1976 are currently found in the New York Bight, and that this region is now being monitored by NMFS scientists.

In summer 1985 an extraordinary brown-tide occurred simultaneously in Narragansett Bay, Long Island coastal embayments and Barnegat Bay - a mesoscale event. The causative factors remain unknown. The causative organism, <u>Aureococcus anophagefferens</u> was previously unknown to science, even the genus. Enormous die-offs of mussels and scallops occurred. The Long Island embayments have been particularly impacted, where this toxic bloom has re-occurred each summer since 1985. The revenue loss to date has been about $10 million.

In mid-October 1987 an anomalous, toxic bloom of <u>Ptychodiscus brevis</u> (the organism implicated in the dolphin die-off) occurred off Cape Hatteras. Paralytic shellfish poisoning occurred and 50% of the scallop population died. An estimated $25 million revenue loss was incurred by the fishing and tourist industries.

Clearly, these representative examples indicate that the coastal waters of the U.S. are likewise exhibiting an increased incidence of nuisance phytoplankton blooms, carrying serious revenue loss and health hazard problems.

There is presently considerable scientific alarm, confusion and uncertainty regarding the nature, causes and regulation of the global epidemic and spreading of nuisance phytoplankton blooms. This partly reflects the historical, scientific approach to treat such blooms as rogue blooms, restricting their investigation to superficial anecdotal descriptions of their occurrences, organisms, general comments on associated environmental/climatologic conditions, and with emphasis on more sensational aspects: spectacular marine animal die-offs; human illness and death resulting from paralytic shellfish poisoning; anoxic outbreaks and development of odorous H_2S; remarkable water discoloration displays; bioluminescence. The literature is packed with such reports.

Our reliance on the anecdotal-rogue bloom approach has led to our inability to explain the causes of the global, nuisance bloom outbreaks; to predict outbreak locations and periods; to account for the spreading phenomenon; to explain the sudden transformations of benign species into toxic ones; to account for local outbreaks, etc. It has also led to our failure to formulate sorely needed testable hypotheses upon which to design much-needed field and experimental research into nuisance blooms. This has tended to perpetuate the anecdotal approach to such blooms. Equally important, this situation has precluded scientifically sound debate and inquiry as to the extent to which the global epidemic of such blooms is primarily an anthropogenic event or triggered by natural, long-term variability and trends in climatic and hydrographic patterns. And, if primarily anthropogenic, what factors are specifically responsible generally, and for a given region.

A striking aspect of the nuisance bloom epidemic is its co-occurrence with the well-documented planetary trends in and stresses of acid rain; the "greenhouse" effect; increased UV irradiance accompanying ozone layer destruction; deforestation; changes in riverine nutrient loading and delivery to coastal environments; and coastal eutrophication. Associated with each of these global patterns are changes in growth factors which may influence bloom dynamics: nutrients; temperature; CO_2 buffering; irradiance; trace metals. Is there a linkage between nuisance blooms and these other planetary trends and stresses? We can not even begin to address this first-order question until we have a better understanding of nuisance bloom phenomena.

It seems clear to me that we have an ongoing equivalent of a "silent spring" in the sea, and that such parallel catastrophies as the dolphin die-off are a further manifestation of this aberration and must be viewed in this context. Consider also the fact that phytoplankton have occurred in the sea for more than 3 billion years, where they have evolved, adapted, regulated biogeochemical cycles, and have served at the base of marine foodwebs. The resilience of this remarkable group of photosynthetic, microscopic algae is well known, i.e., their ability to tolerate environmental assaults and stress. Is the increased global frequency of their anomalous bloom dynamics; the greater, emergent significance of nuisance species, and associated ecosystem dysfunction an indication of their loss of resiliency? That is, should we consider such events as the ultimate "miner's canary"? That the dysfunctioning of this ancient, but major biotic component of Planet Earth is a particularly notable symptom that our planet and its ocean are being pushed to its ecological limits prior to even more serious dysfunction?

I am hopeful that the Committee on Merchant Marine and Fisheries will find this information useful, within their purview and interest; that it will evaluate this matter further, and then submit appropriate legislation designed to better understand and to remedy such deterioration of our ocean, its biota and ecosystem. Thank you.

MAY 02 '89 16:29 I N R S R RIMOUSKI 418 723 7234 P.2/4

WRITTEN STATEMENT

On the mass mortality of bottlenose dolphins
on the U.S. East Coast in 1987

To the

US House of Representatives
Committee on
Merchant Marine and Fisheries

by

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May 1989

The following is a summary of two annexed documents, dated September 1987 and March 1989 respectively, that I have written on this issue. Having not examined any of the dead dolphins nor their tissues for lesions and toxic residues, I am simply responding to statements from and to an investigation report prepared by the team headed by Dr J.R. Geraci.

Basically, investigators concluded that the die-off event was due to intoxication with a natural biotoxin produced by red-tide algae. My evaluation of their report :

1) challenges this conclusion on the basis that the evidence given is weak and at best circumstancial;

2) points out that additional information on lesions and potential chemical agents in tissues is lacking;

3) urges that alternate scenarios be evaluated.

1. It has not been demonstrated that brevetoxin was the causative agent.

- brevetoxins are difficult to quantitate (lack of reliable reference standards, lability of toxins under extraction procedures, loss in preparative steps);
- there is no convincing evidence that the dolphins did indeed have access to sufficient amounts of contaminated fish;
- there is no clinical data on the specific effects of acute or chronic exposure to brevetoxins;
- there is nothing in the literature to suggest that brevetoxins cause chronic liver lesions and immunosuppression, both of which were prevalent in the dead dolphins.

2. Evidence is lacking on lesions and chemicals.

- some types of lesions that would normally be found in such a large collection of dead animals (and I cite tumors as an example coming readily to mind) are not mentioned;
- some organs are not reported on from many, if not all animals;
- no data are given on many chemicals of known toxicity (for example: PAHs, dioxins, furans);

- the results of organochlorine analyses are not very informative in view of the present state of knowledge on such chemicals. In particular,

  a) the discussion, specifically with regards to controls, does not fully consider age differences between animals (to which concentrations of chemicals are related);

  b) it is now known that most of the toxicity from PCBs may come from a few coplanar congeners (namely Nos 077, 126, 169) which are structurally similar to dioxin and behave in the same way. The report does not give any information on these congeners.

3. Other scenarios should have been evaluated.

The most remarkable shortcoming of the report is that

1) with so little hard evidence implicating brevetoxin,

2) while in the presence of remarkably high levels of chemicals of known toxicity, namely organochlorine compounds such as PCBs and DDT,

3) and with much evidence in dolphin tissues of the effects (for example, immuno-suppression, chronic liver lesions) known to result from exposure to these very chemicals,

it fails to suggest an alternate scenario to the one involving brevetoxin. It would have been very plausible to suggest that, as a result of stress induced by some initial injury, dolphins would have intoxicated themselves when reclaiming blubber reserves loaded with organochlorines accumulated through years of living along a contaminated coast.

The search for the initial cause triggering such a chain of events is still open. True, it may be exposure to a natural toxin such as brevetoxin (thus reversing the respective roles of brevetoxin and organochlorines as suggested in the report), but several other agents should be investigated.

In conclusion, I believe that we are still in doubt as to what exactly happened along the Eastern seaboard in 1987.

Pierre Béland
May 2, 1989

NOTE: This is a review of the January, 1989, edition of Dr. Joseph Geraci's final report on the investigation of the 1987-88 mass mortality of bottlenose dolphins.

CONFIDENTIAL

MASS MORTALITY OF BOTTLENOSE DOLPHINS :

Review of the Final Report by J.R. Geraci

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SUMMARY

The research team ascribed the mortality of over 740 dolphins to intoxication by a natural biotoxin, brevetoxin. This toxin had its source in the Gulf of Mexico, far from the area of distribution of the population of dolphins that suffered mass mortality. Only 8 of 17 dolphins analyzed for the toxin tested positive for it.

The report fails to present analyses of toxic organic chemicals other than those that have been traditionally measured in marine mammals worldwide for the last two decades (basically PCBs and DDT). These compounds, the toxicity of which has been well established in other mammals, were found at remarkably high levels in the dead dolphins. However, the report glosses away the role that these very compounds may have played in the demise of the dolphins.

The discussion of pathological findings leaves much to be desired. The array of chronic disorders observed in the dead dolphins, particularly involving the liver and respiratrory system, is explained through generalities regarding assumed effects of brevetoxin. No completely satisfactory explanation is given for the remarkably frequent loss of epithelium from pulmonary bronchioles. Nor is one given for the commonly observed lesions of the skin (excluding the cases of pox-type disease), snout and mouth. Also, the list of pathological findings appears to be incomplete; for example, no data are given on the incidence of tumors in the stranded dolphins.

In its present form, the report is far from convincing. In fact, the author has chosen to write it in such a way that he appears to be trying to convince himself. Alternate scenarios should have been considered, and more investigation is required to satisfactorily establish the cause of the mass mortality.

PATHOLOGY

The treatment of pathological findings leaves much to be desired.

Skin lesions

What seemed striking from an earlier assessment of the strandings (see Béland, 1987, unpublished report) were the numerous skin lesions. Commonly, there were blisters, craterings and ulcers over the head, particularly on the lips, snout and soft tissues of the mouth. There was also sloughing of the skin over large areas. The Geraci report describes these briefly, but fails to provide a satisfactory explanation.

As stated in the report, the primary cause of these lesions does not appear to be viral. Unfortunately, Table 2 on viral results does not include skin analyses. The following summary was extracted from the text, which is not always clear. Skin examinations did not reveal any retrovirus; Herpes-like particles were recovered from a single mouth lesion; and apparently one reovirus-like form was found in a few (?) mouth lesions. The latter is suspected of being a subordinate pathogen. In addition, there were a limited number (only 8) of apparently easily diagnosed cases of dolphin pox.

The report states that the primary cause of the skin lesions was not bacterial either. There is however but one short paragraph on bacteriology in the Results section, while Table 5 again does not list findings in skin and mouth tissues. Most bacteria isolated are opportunistic forms, dominated by the Vibrio group. The discussion section states that bacterial infections were secondary infections (bacteria 'seemed to have been associated with some of the problems in skin and blood vessels that ultimately killed many of the animals but did not appear to be the primary cause of disease' (p.16). Immediately following this vague statement, the report concludes that 'the overwhelming nature of some of the infections, which probably arose in the lung, may have been related to immunoincompetence resulting from chronic stress'. It is not easy to evaluate what the expression 'overwhelming nature of some' actually means quantitatively. And it seems that the reader is to conclude that the skin lesions originated in the lung.

An obvious alternate scenario would assume that the skin lesions appeared first. Then, either because the lesions were extensive, or because the animals already had a depressed immune system (or both), they were unable to cope with invading opportunistic bacteria and viruses.

Chronic fibrosis

Chronic fibrosis was observed in various tissues (liver, lung, heart, lymph nodes; see Tables 3,4 pp. 33-34). Such animals had therefore been ill for some time, as was evident in the first animals that drifted to Virginia Beach in late summer 1987 (p.15). Several animals dying later showed severe hepatic lipidosis, hepatocellular anisokaryosis and single-cell necrosis consistent with toxic hepatopathy.

A whole array of chronic disorders including fibrosis of the liver and lung, adhesions of abdominal and thoracic viscera, as well as myocardial scar lesions, is explained through an alleged 'train of events' precipitated by sublethal exposure to brevetoxin (p. 18). Similarly, liver fibrosis is explained through some alleged but undescribed and unknown effect of chronic exposure to brevetoxin.

Alternately, in lieu of brevetoxin, a chemical agent could have been invoked. Indeed, the report states (p.18) : 'We suggest that PbTx, either alone <u>or in combination with other hepatotoxic substances</u>, was responsible for the general pattern of liver disorders.' (Emphasis is mine). Based on levels reported, it is obvious that the dolphins had life-time exposure to toxic organochlorine chemicals.

Respiratory problems

Commonly opbserved respiratory problems were considerable subleural and parenchymal fibrosis, chronic tracheitis, and, in particular, loss of bronchiolar epithelium. Again, brevetoxin is invoked : 'Here there may have been a role for brevetoxin which disrupts pulmonary function directly through its action on neural control of respiration and by inducing bronchoconstriction' (p.18). However, the author of the report notes that the observed lesions 'might have been associated with inhaled irritants or opportunistic viral or bacterial pathogens.' (p. 18). Nevertheless, without providing supporting evidence, the report rules out both, while assuming that a pre-existing disorder (i.e. brevetoxin) would have facilitated viral or bacterial pathogens. This other agent could equally well have a toxic chemical.

Finally, the report excludes a toxic aerosol as a possible cause (p.18), apparently because 'We propose a line of evidence that excludes a toxic aerosol'. Where is that line of evidence (avoiding circular reasoning) ?

Other findings

Unfortunately, the report does not present all pathological findings; the list of lesions (Tables 3 and 4) is incomplete. In particular, there is no mention of tumors. In a previous study of stranded Atlantic white-sided dolphins, Lagenorhyncus acutus, Geraci et al. (1987, Can. J. Fish. Aquat. Sci. 44: 1289-1300) reported 10 tumors in 41 animals. If, as suggested in another paper by Geraci et al. (ibid. 45:1856), this ratio is taken as a standard, then the 298 Tursiops necropsies should have produced some 73 tumors. This, in itself, would have been a finding worth mentioning, the incidence of tumors being a rough index of the health of a population (by the way, I find the ratio of 10 tumors in 41 animals to be rather high for a healthy population).

Bacteriological and virological reports (Tables 2 and 5) do not include analyses of skin lesions (according to Table 1, 721 viral analyses were performed, but Table 3 lists only 631).

TOXICOLOGY - BREVETOXIN

Contrary to saxitoxin, a water-soluble neurotoxin, brevetoxin is lipid soluble. I have no experience with brevetoxin analyses, but I wonder whether the analytical procedures and partitioning characteristics of brevetoxin make the results dependent on the lipid content of the tissue. If so, a liver tissue with a higher lipid fraction may end up showing more toxicity in a bioassay than a leaner tissue. This may however not necessarily imply that one or the other dolphin was more, or less, at risk when it was alive. Table 6 (Results of brevetoxin analysis in dolphin liver samples) should perhaps have included lipid contents of each sample analyzed. It may be that brevetoxin concentration in the liver is related to lipid metabolism. The health status of an animal, and whether or not it was processing lipids form its blubber, may thus influence the toxicity of its liver as measured in a bioassay.

Only eight dolphins, out of a total of 17 analyzed, tested positive for brevetoxin. This is not a large number, and it would have been wise to give (near Table 6) additional information on those eight dolphins : age, sex, lipid contents of tissues, toxic contaminants levels, pathology.

TOXICOLOGY - CHEMICALS

Organochlorines : PCBs and DDT

There is no doubt that the reported levels of PCBs and DDT are very high. They are comparable to or higher than those from other populations of pinnipeds and cetaceans where reproductive and health problems have been reported (see discussion in Martineau et al. 1987, Archiv. Environ. Contam. Toxicol. 16 137-147; and Martineau et al. 1988, J. Compar. Pathol. 98 :287-311). It is true that, with the exception of reproductive dysfunction in seals (Reijnders, 1986, Nature 324:456-457), experimental evidence on specific effects of given organochlorine chemicals is lacking for marine mammals. However, such effects have been well demonstrated in several other mammalian species.

In particular, PCB and DDT levels found in the liver of Tursiops were remarkably high. Such levels may well have resulted from the dolphins reclaiming their fat reserves and associated organochlorine burdens. At the least then, one cannot but raise the hypothesis that, perhaps as a consequence of an initially poor health, the dolphins intoxicated themselves when processing their fat reserves and its associated toxic burden. It would then become a moot point to insist that an event, such as brevetoxin exposure, may have been the cause of the 1987-88 mass mortality. The report goes around this problem with the amazing sentence `Somewhere in the equation we should consider the role of chlorinated hydrocarbons'.

The discussion of organochlorine residues in dolphins of different ages, sex and origin is incomplete. It is well known that OC levels relate to age and sex of an animal. What then were the ages of the captive dolphins, so that a meaningful comparison could be made ? How healthy were those captive animals ? (How healthy are captive Eastern seaboard Tursiops in general; when taken into captivity, do they respond well to disease without a battery of health tests and treatment ?). What were the causes of the deaths of the captive Tursiops that provided liver samples ?

The report tries to make something out of correlations, or lack of, between residues in liver lipids and amount of such lipids (p. 14). I do not follow what the author is trying to show. In any case, I note that (on p. 13) all animals with 15% or more liver lipids were immature; then it is not surprising to find (on p. 14) that none of the animals with 15% or more lipids had 200 ppm or less, as immature animals tend to have lower burdens anyway.

<u>Heavy metals</u>

None seems to be particularly high. I however challenge the results for Cadmium.

<u>Other chemicals</u>

No results were reported for the more toxic organochlorines such as some PCB congeners, mirex, furans and dioxins. Nor are there any results for hydrocarbons, polycyclic aromatic hydrocarbons, etc... Where are the figures showing negative results for radio-active materials ? At the Washington briefing to various interested parties, held at USNMFS on September 10, 1987, the team leader, Dr Geraci, announced that their investigations included `in excess of 40 000 organics, 80 metals, along with addictive drugs'. Where are all the data ?

Finally, bio-indicator analyses may have provided interesting leads.

CONCLUSION

For the critical mind, the report is lacking in data, and many questions remain unanswered. When ascribing the mass mortality event solely to a not well-documented biotoxin event; while admitting that toxic chemicals may have played a role, the report is unconvincing.

Other scenarios could have been proposed. Here is one. The dolphins were exposed to some toxic chemical in the water (and perhaps at the air-water interface) which, through chemical injury to the skin, mouth, lips and blowhole region (and perhaps to bronchiolar epithelium), led to lesions that got infected by opportunistic micro-organisms. Perhaps due to the extent of these injuries, or to the dolphins' high burdens of organochlorines accumulated over years of living in a contaminated caostal area, their immune system was not up to the challenge. Over time, as sick animals were reclaiming their blubber reserves, exposure of vital tissues to organochlorines increased, catalyzing events leading to death. In some animals, brevetoxin may have been an accidental, perhaps complicating, but not essential ingredient. To document this scenario, incidences of sea dumping of chemicals at designated sites (such as 106) in the first half of 1987 would have to be researched. What kinds and volumes of chemical solutions were dumped ? Did ships crews observe large schools of dolphins nearby ?

Several other scenarios could be proposed and evaluated.

P. Béland
March 1989

THE EAST COAST DOLPHIN STRANDINGS OF 1987

A preliminary assessment
based on discussions with the investigators

Contents

UT NATIONAL
ECOTOXICOLOGIE
DU SAINT-LAURENT

ST. LAWRENCE
NATIONAL INSTITUTE
OF ECOTOXICOLOGY

Pierre Béland,
September 15, 1987

310 avenue des Ursulines
Rimouski (Québec)
[illegible]

CONVERSATION WITH BOB SCHOELKOPF

Marine Mammal Stranding Center
Brigantine, NJ 08203 (609) 266-0538

Washington, September 10, 1987

Cases have been reported from New-Jersey to Virginia, starting in New Jersey on June 15, and later further South. All are individual strandings of drifted animals in varying states of preservation. Several died on the beach; one taken to an aquarium died within three minutes.

Strandings occur at any time of day. They all involve the species (Tursiops), except for one Stenella; all these animals had basically the same condition (described briefly below).

The death toll on Jersey (and Delaware) coast was 85, between June 15 (first case) and Sept 8 (latest case). A few hundred more cases have occurred south to Virginia (see section 2).

Animals are of both sexes, and of all age classes.

The common features of gross examinations are : animals present external blistering lesions, skin peelings, ulcerations; congested lungs and blowhole lesions are often found; the animals give a strong foul smell, probably due to the abundant necrotic tissue; there was some internal abdominal growth on digestive tract. Stomachs were generally empty; when food was present, it consisted of clumps of bones. There were no abnormal injuries or scars, except for an overabundance of soft-shelled barnacles, and of their scars. Overall, dolphins were not emaciated. Parasite load was perhaps smaller than usual; a few animals had tapeworms.

A number of animals were examined and necropsied; all major tissues were sampled for histopathology and toxicology. There are no results out yet. Relative to animals examined further South by Dr J. Geraci, those examined by the Schoelkopf team had possibly more acute lesions, with less fluids; however, gross descriptions fit the same picture.

Potential environmental causes: In 1987, summer was warmer than usual; Gulf stream eddies moved right up to coast, bringing clear southern waters to within four miles of beaches, and potentially holding coastal waters closer to shore for longer periods than usual. No unusual occurrences of algal blooms or red tides were reported. There was an apparently normal supply of food around for dolphins. Reports of chemical spills in the region included xylene and hexane (but tests proved negative), and ozone. The latter was apparently related to a significant number of people reporting breathing problems to local hospitals in early summer, which is believed to be worth investigating further.

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BRIEFING BY J. GERACI TEAM
US National Marine Fisheries Service
Washington

Washington, September 10, 1987

As of August 5, 1987, a US interdepartmental team, headed by Dr J. Geraci of the University of Guelph (Ontario) has been investigating the problem of dolphin strandings. A number of experts from various hospitals, universities and US governmental agencies are involved.

The Smithsonian stranding programme, headed by Jim Mead, has so far recorded 375 Tursiops strandings between June 15 and Sept 10, along the East Coast, from New Jersey to Virginia. This is far above the usual number found per year.

Also, at Virginia Beach, one Stenella and one large offshore Tursiops were found; the first one had the same condition (described below) as all other Tursiops; the second one had a mild (?) condition of the same type.

Sex ratio is 1:1. There are animals of all sizes and ages. The age distribution of strandings is thought to be representative of a cross section of the population. Sample is long on animals in the 5-15 year age groups, and short on old animals; there were a number of calves. The smallest carcass was slightly over 3ft long, which is below the average size at birth. Some females were pregnant, others were lactating.

The bulk (90%) of strandings occurred in waters adjacent to Hampton, Norfolk, Virginia Beach, and immediately South. Some were found in Chesapeake Bay, 40 mi. from Norfolk. Aerial surveys of beaches further South (towards Cape Hatteras) reported no strandings.

Dr Geraci's first examinations revealed extensive skin peeling and ulcerations. These led him to first look for Vibrio's, which are opportunistic microorganisms commonly found in the sea, that may become active and infect animals. The first few necropsies and microbiological analyses revealed that the animals had died of generalized infections and septicemia. Several bacteria and viruses were involved, representing species that are readily available in the environment and opportunistically invade and infect diseased animals. Investigators did not appear to be faced with a uniform problem: some animals showed acute cases, having died within days of infection; others had apparently lingered on for weeks. It seemed that all animals had been weakened prior to invasion by the microorganisms responsible for the obvious lesions. They had been weakened either through immunosuppression, or through some malfunction in a major organ. There were no clues as to what primary cause had rendered opportunistic microorganisms so efficient.

Eventually, a clearer, although not more certain, pattern emerged. It appeared that animals were dying of various conditions :

- some were dying of a skin disease, apparently of viral origin, e.g. a dolphin pox type disease.

- others clearly did not have this condition at all. They showed repeated episodes of various systemic infections from which they would have become weakened, and eventually died.

Animals had an abundance of dark wine fluid (there is some indication that those with more fluids had less acute conditions). None of the animals were robust (whereas early New Jersey animals were). Already in early August, most animals had little food in their stomachs. The last animals to be found were very thin.

To summarize, the dolphins are dying of massive infection of the whole body, of blood vessels (with formation of emboli), with ulcerations and loss of the top skin layer. The internal organs are also receiving similar insult, all apparently from a whole range of microorganisms (including even mycotic pneumonia), many of which are normally found in their environment. Further investigations may reveal whether a single or more species other than those identified so far, or particulalry potent strains of the commonly found species, are involved.

Parasite load is normal. There is no evidence of unusual penetration of skin. A number of animals have soft-shelled barnacles on their backs. Dr Geraci suggested that when such external parasites are found in large numbers, it indicates that the dolphins have been moving slowly. That some barnacles are large may indicate that the problem has been going on for some time.

Microbiological investigations include a wide spectrum of anaerobic and aerobic bacteria, of viruses and of fungi. So far, in excess of 55 *Vibrio* spp., and a number of other opportunists such as *Edwardsellia tarda* have been identified.

Again, there was the suggestion that the animals would have been weakened, stressed or immuno-suppressed in some way by a still unknown primary cause. It is possible that such an event would have occurred some weeks ago and that the bacterial and viral infections sprung from then on. From previous studies on the stress response of dolphins, it is known that different patterns of hormonal response are involved relative to seals. Dolphins generally do not mount a strong response to infection, and are therefore liable to succumb to significant sources of stress.

Primary causes under investigation include :

- competition for food (lack of food would weaken animals);
- biotoxins, or natural toxins (e.g. jellyfish or algal toxins);
- chemical contaminants.

Answers so far are limited :

- There were a few fish kills on the East Coast, but only of a small estuarine scale;
- No extraordinary phytoplankton blooms have been noted;
- There is some indication that a large warm water ring would have come close to the coast in early summer, dissipating slowly in early August. There were some reports of very extraordinary landings of southern fish, but the overall data do not support any significantly large change relative to previous years.

A battery of natural and man-made chemicals is being investigated in various tissues of dead animals, in what the team calls "as complete a search for toxicants as one can think of - in excess of 40,000 organics, 80 metals, along with addictive drugs". The team is also examining the recent discharge story along the coast.

Overall, it looks as if some event could have happened somewhere North (e.g. near New Jersey) in early summer. This event would have been responsible for early deaths in less resistant animals, which turned up on New Jersey beaches. Animals presently beaching in Virginia would be those that survived longer.

With the help of Seaworld personnel, three wild _Tursiops_ were captured alive within the area suspected to support the affected population, in order to investigate the condition of live dolphins in the area. All three had some element of a skin condition; one had abnormally high amounts of fluid in the chest cavity. None was perfectly healthy. Blood samples were taken. All animals died shortly after capture.

It is not known whether the _Stenella_ and offshore _Tursiops_ faced with the same condition had developed it after having come closer to shore, and presumably in contact with the other _Tursiops_, or if they had developed their condition offshore.

Finally, the possibility that we may be looking at some "normal" cyclical epizootic disease event was raised.

The team is also investigating what this event will mean for the East coast dolphin population as a whole. First, there is a need to assess the extent of mortalities. When the wind is westerly (from land), no animals are washed ashore; when the wind is easterly, strandings resume. As dead dolphins first tend to sink and will resurface only when bloated, a number can be lost. Sharks in coastal areas may also take weak or dead animals. It is therefore believed that the count of 400 or so animals is a minimum estimate of the total number of deaths that have occurred. "Conservative" team figures are around 1000+ dead _Tursiops_. It is known that _Tursiops_ can be found both inshore and offshore to 120 nautical miles. Standard sampling surveys were (and will be) carried out, to compare eventually with figures from the 1970s and 1980s. Recent surveys reported 1200 animals very near shore. Overall, North of Cape Hatteras, it is estimated that there are presently 6,000 to 8,000 animals.

Future plans:

- chemical analyses of tissues for toxicants and contaminants; .
- capture of more live animals to evaluate stress condition from hormonal analyses, for matching of antibodies with known pathogens; evaluation of functional condition of kidneys, liver and other major organs.
- histopathology of samples collected so far.

It is expected that any answer will be long (months) to come. No findings will be released before thorough checking.

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OVERVIEW AND DISCUSSION

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Caveat : What follows is based on the limited information available from the above conversations. None of the animals or samples have been seen by members of our staff.

The condition of generalized infections by a variety of opportunistic microorganisms, as described above, undoubtedly supports the interpretation that the defense mechanisms of the stranded animals were abnormally low. This however, as stated by those who have examined the carcasses, cannot however be a definitive answer.

First, it must be emphasized that all we have so far is strong circumstancial evidence of immunosuppression. There is no direct analysis showing that the animals were indeed not mounting a reasonably strong defense against infection. I suspect that the capture of live animals may help to clarify that.

Secondly, should immunosuppression be indeed involved, the primary cause that has put the dolphins in such a state still remains to be determined.

A priori, from an evolutionary point of view, it would not make sense to simply suggest that dolphins in the wild in general do not mount a "strong" defense against infection.

Without any past evidence at hand, it is hard to comment on the possible occurrence of cyclical "natural" epizootics. I believe that a search through stranding files worldwide would suggest that there is little evidence for them.

In this context, a thorough search for a primary and, relative to strandings in previous years, unusual cause is essential. So far, investigators and concerned individuals have suggested :

- natural biotoxins, such as that from red tide algae;

- anthropogenic toxicants (pollutants), either in the water, sedimen ., food or atmosphere (ozone), including radio-active material;

- viral or bacterial disease - as a distinct species or a particularly virulent form of the opportunistic strains already identified.

On the basis of available information, none of these can presently be confirmed nor rejected, but future research ought to eliminate some.

A search for a primary cause should include considerations of space, time and specificity :

Space : The suggestion that some element of stress (in the broadest sense) was encountered nearshore by those dolphins during their annual migration North appears to be a promising first lead. However, the two Stenella and one offshore Tursiops also found with the same condition suggest that the source of the problem may not be easily located within a small area.

To help substantiate the hypothesis of some event happening somewhere in the North in early summer, a comparative analysis of the conditions of all animals found at all latitudes throughout summer should be done. Can it be ascertained that there was an evolution of the condition of animals from North to South ? Were the animals dying in August in the South indeed survivors of an event that occurred in the North, or has the causing agent also "moved" South ?

Time : How much time is required for strains of opportunistic organisms to cause the types and extent of lesions and ulcerations found on beached dolphins ? Can the facilitation of barnacle attachment by slower moving dolphins be substantiated ? If so, an evaluation of barnacle growth rates would provide an estimate of the earliest occurrence of a causative event. If the culprit is a contaminant, we are dealing with acute (as opposed to chronic) poisoning; what types of potent chemicals were available then in sufficient concentrations to affect so many animals ?

It should not be ruled out either that the causative event may have occurred several months before the first deaths were recovered.

Specificity : The search for a primary cause may be long and costly. It would be interesting to know more about the strategy being followed by Dr Geraci's team for finding, out of an overwhelming 40,000 plus possibilities, the likely major toxicants, if any, in tissues of deceased (and live) animals. A first series of analyses on a few animals, along with attempts to match symptoms with known effects of such contaminants, and a description of possible sources (in space and time) of such contaminants would help in defining promising directions for further research. An initial search should concentrate on anything significantly above background levels.

At the same time, toxins and contaminants should be looked for in other biota, specifically those in the dolphin diet, within the time and space frame corresponding to and immediately previous to the occurrence of the first strandings.

The presence of two Stenella among strandings, and any difference in their specific response to the causative agent, may help in accepting or ruling out the epizootic hypothesis.

Strandings are often selective relative to size nor do recorded deaths account for total mortality from a population, particularly over an area as open and as wide as the Eastern seabord. Based on survey estimates, and on the number of deaths so far, there is no doubt that the event has affected a very substantial proportion of the inshore East Coast Tursiops population. It is essential to evaluate what this means for the future of the inshore Tursiops population, as well as for other populations. At present, there are two groups, one in the North, one in the South, that have done necropsies, bacteriological and chemical analyses independantly. To answer some of the above questions, collaboration between the two groups will be necessary.

Pierre Béland,
Science Director,
September 15, 1987

STATEMENT OF
GABRIEL A. VARGO
DEPARTMENT OF MARINE SCIENCE
UNIVERSITY OF SOUTH FLORIDA
ST. PETERSBURG, FLORIDA

BEFORE THE

SUBCOMMITTEE ON OVERSIGHT AND INVESTIGATIONS
U.S. HOUSE OF REPRESENTATIVES

MAY 9, 1989

Mr. Chairman and Members of the Committee:

My name is Gabriel Vargo and I am an Associate Professor at the Department of Marine Science, University of South Florida. Thank you for the opportunity to address this committee.

Introduction

We have been asked to consider several aspects of the report submitted by Dr. J.R. Geraci for NOAA on the mass mortality of the bottlenose dolphin, *Tursiops truncatus*, along the east coast of the U.S. during 1987-1988. I will attempt to address each of the areas specified in your letter as separately as possible although some overlap is inevitable.

My review of this document can be summarized with the following comments:

1. Dr. Geraci and his associates should be recognized for their efforts in organizing and co-ordinating the multi-state team of scientists and laboratories required for this study and for their foresight in considering the possiblity of biotoxins in their suite of analyses. There is little precedent for this

in studies of marine mammal deaths.

2. The proposed scenario for the involvement of brevetoxin in the chain of events that led to this mass mortality of dolphins is plausible and feasible. Unfortunately the number of analyses upon which this scenario is based leaves it open to question. Additional analyses of stored samples for Pbtx-2 and other toxins or their degradation products should be done to enhance or negate this hypothesis.

3. The finding of, in Dr. Geraci's words, "unprecedented" high levels of DDE and PCB's in the blubber and liver of this coastal species of dolphin is indeed a sad commentary on the state of the environment along the eastern U.S. shore. The dolphins did not accumulate these compounds overnight. Exposure had to be chronic. We should ask the question: Would this mass mortality have occurred if these compounds had not been present?

4. The presence of the toxic dinoflagellate, _Ptychodiscus_ _brevis_, along the east coast of Florida and into North Carolina is an established fact. The presence of the toxin in menhaden, a filter feeder capable of removing phytoplankton in the size range of _P_. _brevis_ directly from the water column, has also been established with this report. Red-Tide blooms of _P_. _brevis_ have been considered, with a few exceptions, as a Gulf

of Mexico phenomena. The 1987-1988 North Carolina bloom and the possible involvement of this organism and terrestrially derived pollutants in the deaths of top carnivores emphasizes that we cannot continue to think of our marine ecosystems as isolated regions, applying statutes and restrictions in one area and not another. Coastal and oceanic waters are a continuum. Events that occur in one region will affect another. In this particular case, any future studies should encompass the entire system.

Additional comments on specific areas of inquiry follow.

A. Methodology

I cannot comment on the detailed methods used for each type of analysis since they are outside my area of expertise. For the purpose of this statement I assume that the numbers are accurate. My assumption is based on the following examples. The laboratories and personnel involved have a history and an expertise in such analyses. Additionally, the biotoxin samples were run as blind tests. This enhances their reliability. Furthermore, all controls were negative. Concentrations of DDE, PCB and lipids were confirmed by independent analyses and concentrations of these compounds in control animals were consistent with published values.

B. Peer Review

I cannot comment on the peer review process without knowing if reviews were done "in house" or by outside reviewers or without seeing their remarks.

C. C nclusions

The scenario presented by Dr. Geraci for the sequence of factors leading to the deaths of unprecedented numbers of dolphins is plausible and feasible although one area open to question is the extent to which brevetoxin was the mitigating agent. Dr. Geraci does indicate that the evidence for brevetoxin is circumstantial (p.19, paragraph 5) and that mobilization of stored PCB's and organochlorines played a role in further debilitating the dolphin populations opening the door to other clinical symptoms which were the immediate cause of death.

Finding brevetoxin in 8 of 17 liver samples indicates that further substantiation of its involvement is required. However all the controls were negative for Pbtx-2 and the toxin could be stored in other organs, flesh or blubber. The presence of toxin in two suckling calves suggests mobilization of the toxin from lipid-rich tissues. This is the first report of brevetoxin in dolphins. To the best of my knowledge we do not know how marine mammals handle biotoxins, where they may be stored, how long they could remain in their bodies, how they may be metabolized or what concentrations yield a toxic response. Furthermore, the analysis was only standardized for Pbtx-2. P. brevis produces other toxins. Degradation products of the other toxins and Pbtx-2 would be recorded as negative. Five additional dolphins tested positive in all three bioassays and displayed a peak in HPLC analysis; including 3 "control" animals. Were these peaks a secondary toxin or a degradation product? This should be determined. The potential involvement of brevetoxin, while intriguing, requires additional substantiation.

Finding brevetoxin in the visera of menhaden, a primary as well as an indirect food source for the dolphins, is also unprecedented. Normally intense blooms of *P. brevis* yield massive fish kills. Thus, earlier research has focused on cell concentrations that produce mortality. I do not know of any analyses that document accumulation in fish at this trophic level due to chronic exposure to low *P. brevis* cell concentrations. This should be done, at least initially, on Gulf coast populations which are frequently exposed to red-tides. Similarly, toxin analyses of samples from dolphin strandings on the west Florida coast would also help clarify some of the questions raised by this report.

Could the enhanced levels of PCB's and organo-chlorines have been the primary agent responsible for the dolphin mortality? The report does consider this possibility although the agent that would yield mobilization of these compounds from storage in the blubber (other than brevetoxin) was not identified. Possibilities include a lower than normal food supply along the migration route and/or a greater expenditure of energy during migration requiring use of reserve fat in the blubber. I suggest that the question that should be asked is whether these deaths would have occurred if the dolphins had not accumulated such a high body burden of pollutants.

There is also a question of timing in this event. Populations of *P. brevis* did form blooms in North Carolina in the fall of 1987; that is established. Yet dolphins were found dead in Virginia 3 months before the bloom in North Carolina. The arguments presented regarding low population levels of *P. brevis* going undetected in the water column have counterparts on the West Florida Shelf. Unless population levels are high enough to yield fish kills, they are seldom detected without a sampling program specifically designed to monitor for their presence.

It is my opinion that _P_. _brevis_ cells are transported from the Gulf of Mexico to the Florida Current whenever Gulf populations are present. Filaments of the Gulf Stream that reach nearshore waters along the southeastern states also occur. It only remains for the proper physical conditions to develop in nearshore waters that concentrate cells and maintain populations in a discrete area long enough to produce a bloom. _P_. _brevis_ does possess the physiologic and biochemical attributes that allow it to persist and grow in the nutrient poor (oligotrophic) waters of the Florida Current and Gulf Stream.

D. Suggestions for additional studies

These suggestions are not listed in any order of priority.

1. Physical oceanographic studies designed to determine the relationships between water movements in the Gulf of Mexico, the Florida Current and the Gulf Stream with particular attention to interrelationships with coastal waters. Several studies have been done in the past. These should be identified with additional research on contiguous areas.

2. Analyze additional samples, including other tissues, from the dolphins stranded during this event for Pbtx-2, other biotoxins and their degradation products.

3. Analyze tissues from dolphins and other cetaceans stranded on the West Florida coast, an area of episodic red-tide events.

These analyses should also include fish that constitute the dolphins food supply and other herbivores.

4. Initiate laboratory research programs on the effects of and accumulation of brevetoxin under conditions of chronic, low cell concentration exposures.

5. Increase the data base on concentrations of pollutants such as PCB's and pesticides in marine mammal populations, in both coastal and offshore species.

6. Enhance our knowledge of migratory routes, patterns, social behavior, the physiological requirements and food supplies for all species that exhibit migrations through a variety of environmental zones. Perhaps it would be possible to identify a species that could act as a "miner's canary" with respect to potential hazards or future problems.

ANALYSIS OF CETACEAN STRANDINGS ON THE ATLANTIC COAST OF THE UNITED STATES, 1978-1988, WITH REGARD TO MASS MORTALITIES OF BOTTLENOSE DOLPHINS DURING 1987 AND 1988

Testimony Provided to the
U.S. House of Representatives
Committee on Merchant Marine and Fisheries
Subcommittee on Oversight and Investigations

by

Melvin H. Goodwin
Andrew S. Mount
South Carolina Sea Grant Consortium

and

Albert E. Sanders
Charleston Museum

During the summer of 1987, large numbers of dead or dying bottlenose dolphins (Tursiops truncatus) washed ashore along the east coast of the United States from New Jersey to North Carolina. By November, similar strandings had been reported in South Carolina, Georgia, and Florida, and by the end of September 1988, 834 bottlenose dolphin mortalities had been documented by the United States East Coast Marine Mammal Stranding Network.

Bottlenose dolphins are among the species of marine animals that are close to the top of the ocean's complex food web, and the observed mass mortalities may be symptomatic of more profound events taking place in other parts of the marine environment. Because humans also consume portions of this food web, concern for the health of the seafood eating public warrants careful examination of the potential causes of the dolphin strandings.

In response to inquiries from private citizens concerned by the strandings and their implications for marine water quality, we undertook an examination of the entire body of Network stranding records from Maine to Florida from 1978 through September of 1988. The purpose of this investigation was

> to assess the scale of the Tursiops mortality in relation to past strandings
> to determine whether similar increases in stranding levels had occurred among other species of cetaceans
> to determine whether the 1987-88 die-off was a true anomaly or if it was an explosive peak in a trend of increasing bottlenose dolphin mortalities within the past decade.

Stranding data from January 1976 through September 1988 were obtained from the Smithsonian Institution, Washington, DC. Data were provided from the Scientific Events Alert Network (SEAN) for the period from 1976 to 1981 and from the Marine Mammal Events Program for the period from 1982 to September 1988. Additional data were obtained from the Southeastern United States Marine Mammal Stranding Network (SEUS) for the period from January 1988 to October 1988.

Information from a total of 2984 cetaceans that had stranded and died on the east coast of the United States from January 1978 to October 1988 was transcribed into a relational data base. Data included stranding locality and date, sex, length, condition, and network serial number. Only those animals that had been positively identified to species level were included in subsequent analyses.

A total of 33 species were represented among the strandings for the period examined (Table 1). While these figures are obviously incomplete because they do not include animals that came ashore in remote areas and consequently were not detected, they provide an indication of the relative volume of cetacean strandings that have occurred in that region within the past decade. Because a central aspect of this investigation involved comparing strandings during 1987 and 1988 with those reported in previous years, we were concerned that app~~arent va~~riations in stranding frequency might be the result of variations in observation effort. If this were the case, one would expect a correlation between the number of cetacean sightings and the number of strandings reported. A linear regression comparison of sighting and stranding data from Florida (1) reveals no such correlation (R= 0.50), suggesting that the number of marine mammal strandings was independent of sampling effort during that period. We have assumed that this result is generally applicable to the data examined in this study.

Mass strandings of many individuals are regularly reported among some cetacean species, particularly pilot whales (Globicephala macrorhynchus G. melaena), sperm whales (Physeter catodon), melon-headed whales (Peponocephala electra), false killer whales (Pseudorca crassidens), and the Atlantic white-sided dolphin (Lagenorhynchus acutus). Mass strandings typically involve all or part of a single herd that comes ashore at the same time and often in the same place. In these respects, mass strandings are distinct from the sort of mass mortalities reported during 1987 and 1988. Incidents of mass stranding are identified by an asterisk in Table 1.

These data indicate that the number of bottlenose dolphins stranding during 1987 and 1988 was unprecedented during the period in which systematic records of such events have been kept. While about 125 - 250 cetaceans typically strand on the U.S. East Coast per year, nearly 800 cetaceans stranded during 1987 (Figure 1). Although the greatest impact was on bottlenose dolphins, 1987 was also the peak mortality year for harbor porpoises (Phocoena phocoena) Atlantic white-sided dolphins (Lagenorhynchus acutus), and humpback whales (Megaptera novaeangliae) (Figure

2). Since the numbers of stranded bottlenose dolphins greatly exceeded those of other species of cetaceans, analyses of temporal and spatial trends were confined to Tursiops.

Stranding numbers exceeding previous yearly averages began appearing in July 1987 in coastal New Jersey, Maryland, and Virginia (Figure 3). By August, the highest numbers of bottlenose dolphin strandings ever recorded during a single month occurred in New Jersey, Delaware, Maryland, and Virginia. Strandings in these states decreased in subsequent months, but anomalously high strandings of bottlenose dolphins occurred in North Carolina in October, in South Carolina during November and on Georgia beaches during December. The progressive increase in strandings reached Florida in December of 1987 and attained a peak during January and February of 1988. Unusually high rates of strandings continued in Florida coastal waters through May of 1989.

With respect to questions posed at the beginning of this investigation, these data

> establish that the scale of Tursiops stranding mortality observed in 1987 and 1988 was several times greater than in previous years;
> establish that similar increases in strandings occured among several other cetacean species; and
> suggest that the 1987-88 event was highly unusual and is not consistent with any discernable trend in strandings during the past decade.

The offical investigation of the dolphin mass mortality event has concluded the most likely cause to be consumption of fish tainted with brevetoxin from a bloom of Pytchodiscus brevis (one of the dinoflagellates responsible for "red tides")(2). There are at least four serious problems with this explanation.

First, the pathology surrounding previous instances in which brevetoxin has been implicated in deaths of marine mammals is quite different from that observed during the 1987-88 event (Table 2). An outbreak of red tide in Fort Myers, Florida from late January to April 1982 was implicated in the deaths of 41 West Indian manatees (Trichechus manatus)(3). Post-mortem examination of the manatees revealed no signs of lesions on organ systems, but in some cases there was evidence of brain hemorrhage consistent with the diagnosis of neurotoxicity. The condition of dolphins stranded during 1987-88, on the other hand, was unlike any that has been observed previously by marine-mammal workers on the eastern seaboard (2). Most of the dead dolphins shared a variety of pathological abnormalities, including small blisters and pox-like lesions about the head, commonly on the lips and in the mouth; sloughing of large areas of skin; pulmonary congestion and hemorrhage; fibrosis of the liver, lungs, and pancreas, and deterioration of blood vessel walls, permitting leakage of plasma into the abdominal and thoracic body cavities (2, 4).

Second, the Fort Myers episode was accompanied by P. brevis concentrations reaching four million cells per liter, fish kills of mullet and catfish, and toxic effects in cormorants additional to those seen among the manatees. In contrast, no bloom of P. brevis was observed in the Atlantic until three months after dolphins began stranding on the northern portion of the east coast in 1987 (5). Moreover, there were no reports of fish kills (6, 7), nor have we been able to discover reports of symptoms of brevetoxin poisoning in other marine animals, or of human illness that would be expected had contamination occurred on a scale sufficient to produce such widespread effects.

Third, blooms of P. brevis are rather frequent in the Gulf of Mexico, yet no mass mortalities of dolphins (or other cetaceans) have been correlated with these events (8, 9).

Fourth, brevetoxin was found in less than half the specimens assayed for that toxin (eight of a total of seventeen samples). Setting aside reservations concerning the extent to which seventeen samples are likely to be representative of an event involving more than eight hundred stranded individuals, with the potential that thousands of animals may have died at sea (10), an explanation is still lacking for the majority of the observed dolphin mortalities.

Aside from these considerations, there are several other possible causes for the 1987-88 event that do not appear to have been sufficiently examined. Of particular concern (because of implications to other species, including humans) is the possible role of point source pollution. Forty-one percent of Tursiops strandings during the mass mortality event occurred along the coasts of New Jersey, Delaware, Maryland, and Virginia; yet this area represents only 19% of the linear distance from the northern New York coast to the Florida Keys. In addition to numerous point sources of industrial contamination, there are a variety of ocean disposal sites within this area, including those containing sewage sludge, acid waste (11) and chemical warfare agents (12).

Chlorinated hydrocarbons, including pesticides and polychlorinated biphenyls (PCBs), have been confirmed in the tissues of bluefish, striped bass, and other marine animals that are found in the coastal waters of the Mid-Atlantic region (13,14,15,16). The fact that most tissues examined from dolphins involved in the event contained high concentrations of organochlorine residues (2) raises the possibility that deleterious substances received through the food web were at least partially responsible for the observed mass mortality of these animals. In our opinion, the implications of this possibility to other species associated with the same food web, including man, provide ample justification for more intensive investigation.

Finally, a few data that have been made available to us indicate relatively high levels of PCBs in the tissue analysis of three dolphins (#85-87, CWP-263, CWP-267) from the mass mortality event that were analyzed at NMFS-Charleston Laboratory on September 3, 1987 (17). These animals are identified in Appendix I of the Geraci report (p.49 and p. 50) as not having been subjected to chlorinated hydrocarbon analysis.

These circumstances -- the unprecedented extent of the mass mortality event of 1987-88, the reservations concerning the explanation which has been advanced, the slight extent to which other potential causes have been examined, and inconsistencies among offical reports -- prompts us to urge that further inquiry be initiated with broad representation from the scientific and technical community to

> identify potential causes of the mass mortality event that should be considered, and
> apply the diverse technical expertise available within the research, commercial, and governmental community to provide an in-depth evaluation of each of these causes.

We agree with a portion of Dr. Geraci's final statement: "of the need to resolve the growing question of whether contaminants at levels found in the dolphins might have affected their resilience and rendered them more susceptible". We do not agree, however, that analyses presented in this report are sufficient to establish a specific causative agent. In offering this testimony we imply no criticism of those agencies and individuals who have undertaken the difficult task of explaining the 1987-88 dolphin mass mortality, but suggest that the task is not yet complete.

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CETACEAN STRANDINGS, U.S. EAST COAST, MAINE TO FLORIDA, 1978 THROUGH SEPTEMBER 1988

	IDENTIFIED TAXA:	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	TOTALS
	SUBORDER ODONTOCETI												
	Family Monodontidae												
1	Delphinapterus leucas...............	1			1						1	1	4
	Family Phocoenidae												
2	Phocoena phocoena....................	25	31	25	20	34	2	11	10	17	58	9	250
	Family Delphinidae												
3	Delphinus delphis....................	9	5	1	13	11	2	2	19	12	7	1	82
4	Feresa attenuata.....................	1				4	3				1	5	14
5	Globicephala macrorhynchus..........	3	8	4		1	1		9	4	32*		62*
6	Globicephala melaena.................	8	7	2	21*		5	10	8	52*		9	122*
7	Grampus griseus......................	1	6	8	5	8	2		7	4	2	1	44
8	Lagenodelphis hosei..................				17*								17*
9	Lagenorhynchus acutus................	11	8	20	23	15	11	12	12	9	50*	3	174*
10	Lagenorhynchus albirostris..........		1			1				2			4
11	Orcinus orca.........................		1	1		9					-		11
12	Peponocephala electra................							1					1
13	Pseudorca crassidens.................						1			3		1	5
14	Stenella attenuata...................						6		2		2		10
15	Stenella clymene.....................						7				2		9
16	Stenella coeruleoalba................	12	11	3	9		4		13	3	2	5	62
17	Stenella frontalis...................			1		2				2	6	3	14
18	Stenella longirostris................	1					1						2
19	Stenella plagiodon...................	8	3	1	8	1	6	2	1				30
20	Steno bredanensis....................			1						2			3
21	Tursiops truncatus...................	95	79	69	80	116	73	54	104	75	581	253	1579
	Family Ziphiidae												
22	Mesoplodon densirostris..............	1			1			1		2			5
23	Mesoplodon europaeus.................	5		2	3	3	2	1	5	1	2	3	27
24	Mesoplodon mirus.....................							1	2	2			5
25	Ziphius cavirostris..................	4		2	3	1	1			4	1	1	17
	Family Kogiidae												
26	Kogia breviceps......................	22	29	17	31	24	25	9	28	21	7	14	227
27	Kogia simus..........................	7	3	7	3	3	3	1	6	2	4	3	42
	Family Physeteridae												
28	Physeter catodon.....................	4	4	13*		5	5		6	3	2	1	43*
	SUBORDER MYSTICETI												
	Family Balaenidae												
29	Eubalaena glacialis..................		2		2	7	1			1		1	14
	Family Balaenopteridae												
30	Balaenoptera acutorostrata..........	3	6	2	5	2	1	1	4	1	6	6	37
31	Balaenoptera edeni...................	2					1						3
32	Balaenoptera physalus................		3	5	2	8	3	1	1	1	3	7	34
33	Megaptera novaeangliae...............	2	1		1		2	1	5	4	13	2	31
	Totals...	225	208	184	248	255	168	108	250	227	782	329	2984
	Imprecisely determined animals...............	6	5	3	9	5	4	4	6	3	5	8	58
	GRAND TOTAL.......	231	213	187	257	260	172	112	256	230	787	337	3042

* Mass stranding involved

TABLE 1. Cetacean strandings recorded on the east coast of the United States, 1978 through September 1988. From data bank maintained by the East Coast Stranding Network. Figures shown represent actual strandings only and do not include sightings of living animals. "Imprecisely determined animals" are records of badly-deteriorated carcasses or strandings reported by persons unfamiliar with cetacean identifications.

BEST AVAILABLE COPY

Table 2. Comparison of Gross Pathology of Tursiops truncatus 1987-88 from the U.S. East Coast Die Off Event to Trichechus manatus from the 1982 Ft. Myers Florida Die Off Event

Tursiops truncatus(1)	Trichechus manatus(2)
Sloughing of large areas of skin to level of subcutaneous tissue	Animals in good flesh with light to heavy fat deposits
No comparable observations	Stomachs were full, indicating recent feeding. Lower gastro-intestinal tracts were full Watery consistency to the contents of the cecum and/or upper large intestine Presence of ascidians in gastrointestinal tract
Ulcers on the palate, gingiva, lips, tongue, and skin	No ulcers noted
Large volumes of port wine-colored fluid in abdominal and thoracic body cavities Spleens enlarged two to three times normal size Yellow discoloration and emphysema of the pancreas Pulmonary congestion, hemorrhagic infarction, and/or bronchopneumonia Liver abnormalities ranging from severe fatty change to extensive cirrhosis (fibrosis)	Significant lesions seldom observed in any organ system
Brain hemorrhage	Brain hemorrhage

(1) Cassidy, D.R., A.R. Davis, A.L. Jenny, and D.A. Saari., 1988; Pathology of the diseased dolphins; Proc. Oceans'88, Baltimore, Md. pp.812-814.
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Figure 1

Reported Cetacean Strandings, 1978 - Third Quarter 1988, Atlantic Coast

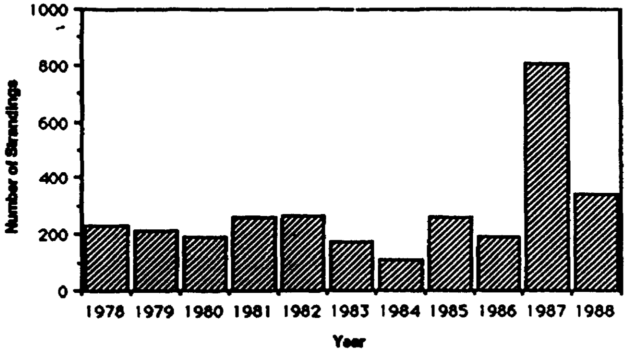
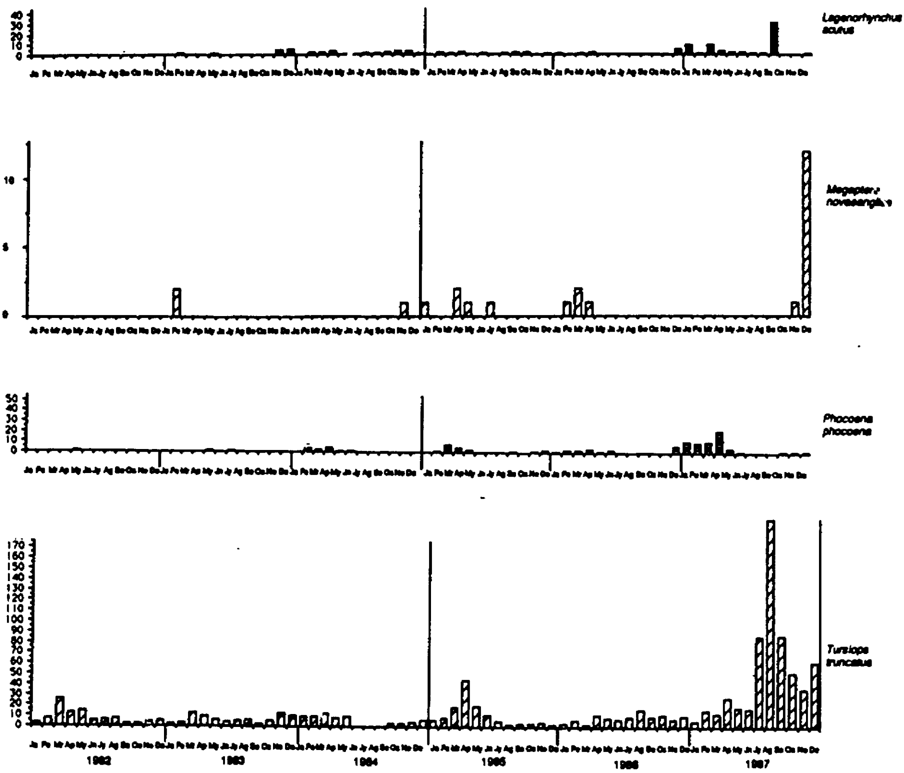


Figure 2

Reported Strandings of Atlantic White-Sided Porpoise (*Lagenorhyncus acutus*), Humpback Whale (*Megaptera novaeangliae*), Harbor Porpoise (*Phocoena phocoena*), and Bottlenose Dolphin (*Tursiops truncatus*), 1982 – 1987



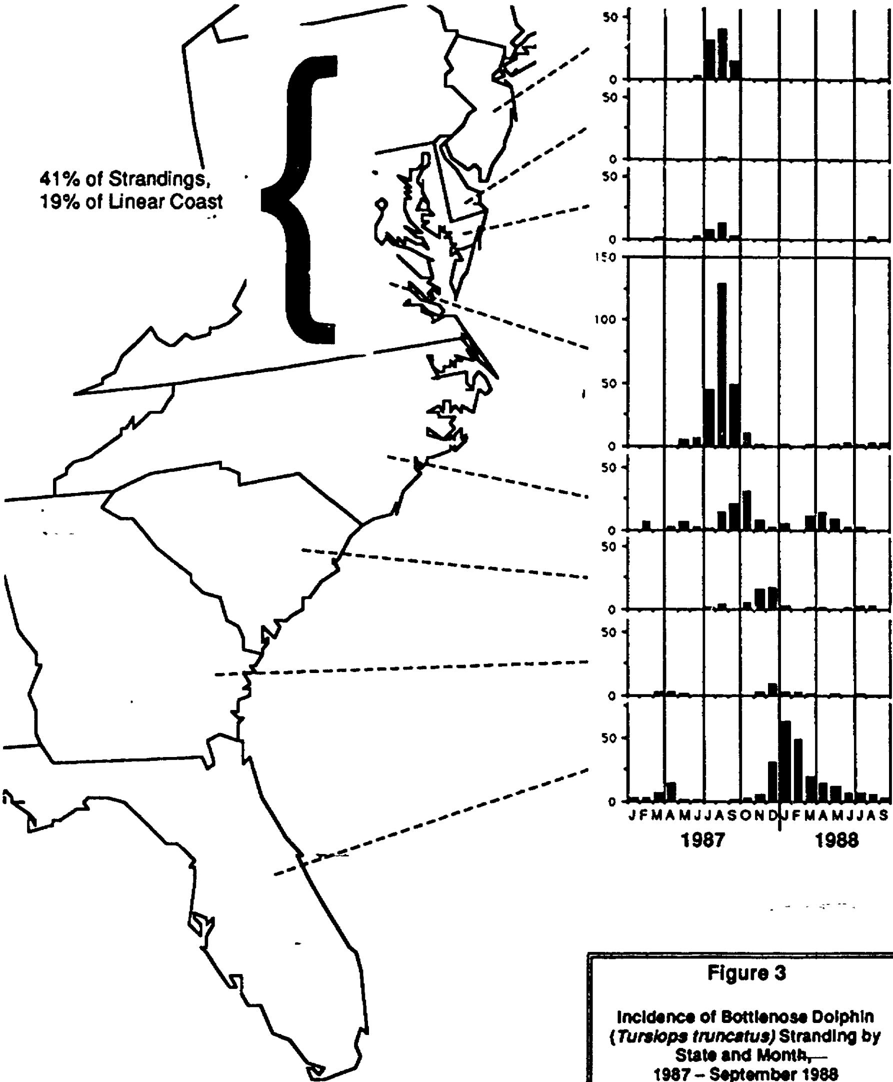


Figure 3

Incidence of Bottlenose Dolphin (*Tursiops truncatus*) Stranding by State and Month, 1987 – September 1988

MASS MORTALITY OF BOTTLENOSE DOLPHINS:

Review of the final report of Dr Geraci

May 7th, 1989

by

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SUMMARY

A report prepared by Dr.J.R. Geraci from Guelph University concerning the stranding of over 740 bottlenose dolphins along the American Atlantic coast (July 1987-March 1988) claims that strandings were caused by a biological toxin (brevetoxin). A compound assumed to be brevetoxin by the author was detected in 8 of 17 carcasses, in one fish contained in the stomach of one dolphin and in 3 fish caught offshore Florida.

However, there is lack of evidence to support that brevetoxin was the major cause of these strandings; the facts support an alternate conclusion that organochlorine compounds (OC) and particularly polychlorinated biphenyls (PCBs) had an important role in the strandings. The reasons for this are briefly summarized as follows: of all the lesions found in the 240 necropsied carcasses, none can be actually related with brevetoxin toxicity since there is no report of the lesions caused by this toxin in animals. On the other hand, many lesions found in the carcasses were described in laboratory and domestic animals intoxicated with PCBs.

High concentrations of PCBs were detected in all carcasses in which analyses were done. Lesions relatively specific for PCBs toxicity such as (parakeratotic) hyperkeratosis were observed on the stranded dolphins. Severe septicemia with a variety of opportunistic bacteria and lymphoid depletion were indicative of profound immunosuppression. PCBs are strong immunosuppressors while brevetoxin is not recognized as such. Yet, the relation of these lesions to high levels of PCBs is ignored in the discussion of this report.

Lesions and immunosuppression caused by PCBs have been extensively documented in laboratory and domestic mammals exposed to levels lower or equal to those found in the stranded dolphins. In contrast, lesions caused by brevetoxin, if brevetoxin causes any lesion at all, are not known; the rare existing studies are concerned with pathophysiological effects (effects on live animals) and potential damages to organs produced by the toxin have not been determined.

Bottlenose dolphins are mammals; as such they have the same basic metabolic machinery as other mammals and are exposed to the same toxic effects. Lesions consistent with chronic PCB toxicity were found in 67 stranded North Atlantic dolphins while high levels of these compounds were found in 53/53 dolphins. These important considerations are absent from Dr. Geraci's complex scenario of brevetoxin-induced events.

Dolphins have been exposed to brevetoxin for thousands of years and, most likely, have developed metabolic pathways to degrade it. By contrast, exposure of bottlenose dolphins to OCs

and particularly PCBs is recent since PCBs are synthetic compounds introduced in the marine environment less than 50 years ago; other mammals, in which OC toxicity has been studied, have not evolved efficient OC detoxifying mechanisms. Most likely, bottlenose dolphins are the same.

INTRODUCTION

Each section of Dr. Geraci's report is reviewed. Excerpts of the report are underlined while my own comments are not. Simple definitions of technical words are included between brackets. An earlier report in which the lesions found in 10 stranded dolphins were described by veterinarians from the National Veterinary Services Laboratories (NVSL), Ames, Iowa, (Annex A) is compared to the final report; there are some inconsistencies between the 2 reports.

PATHOLOGY

GENERAL COMMENTS

While a report of histological lesions from 10 dolphins (Annex A) mentions marked post-mortem changes (autolysis) of many major organs, Dr. Geraci's report does not mention the severe autolysis of carcasses (changes occurring after death hampering microscopic examination of tissues). Not all organs of each carcass were examined microscopically (Table 1). Therefore, failure to report a lesion in a particular organ was not always due to the absence of lesion but rather to the fact that the organ was not examined. For instance, the nervous system is remarkably absent from the results (Tables 3 and 4). Moreover, any particular lesion could have been present in more dolphins than is reported.

For instance, squamous metaplasia of glands is a feature of OC toxicity in mammals and this lesion has been observed in mammary glands of monkeys[21] intoxicated with PCBs. The same lesion, observed in stranded dolphins[10] and in beluga whales[20], was suspected to be caused by PCBs. Examination of mammary glands is not mentioned in the report. Recognizing these limits, the following comments are necessary:

SPECIFIC COMMENTS

GENERALIZED VASCULITIS

The vascular lesions described microscopically in an earlier report, Annexe A, consisted of vasculitis (inflammation of blood vessels) and were present in 9 of 10 dolphins. Moreover, the following comments of Dr. Geraci indicate that this lesion was recognized microscopically and that this lesion was frequent.

Table 3 lists lesions seen with the naked eye. Yet, vasculitis, which can be characterized in details only by microscopy, is listed only in Table 3 and not in Table 4 which lists the lesions observed with light microscopy.

"These lesions were associated with thrombosis of dermal vessels, presumably caused by bacteria, fungi, or protozoa." p. 9.

"Other findings in the dolphins were also related to septicemia, and particularly to the effects on blood vessels which had been injured by bacteria. The vessels walls became fragile and necrotic, and were unable to contain blood. Plasma leaked into tissue spaces causing edema in many of the organs, and accumulation of massive quantities of blood-tinged fluid in the thoracic and abdominal cavities....."
p. 10

I agree with the author on the significance of these vascular lesions (which are not reported in Table 4 of his report): briefly, blood flow was invaded by a tremendous number of opportunistic bacteria (septicemia). This led to inflammation of blood vessels and their obstruction by blood clots. These events alone can explain most gross findings: liquid in abdominal and pleural cavities, edema and necrosis of organs, including skin, which were irrigated by the occluded blood vessels.

SKIN - PARAKERATOSIS

Hyperkeratosis is a thickening of the most superficial skin layer. Parakeratosis is a variant of hyperkeratosis. The author does not comment about parakeratosis which was observed in 24% of the dolphins. Considering that skin from only 6 dolphins out of 10 (Annex A) was examined microscopically and that this percentage was probably the same for the other dolphins, this lesion was most likely present in more animals. This lesion deserves more comments since skin parakeratosis is reported in animals and humans intoxicated with PCBs[23]. There are no attempts to explain this lesion.

Curiously, inflammation of integumentary blood vessels, which can be described in detail only by microscopic examination is reported in Table 3 where gross findings are listed and is absent from Table 4 where microscopic lesions are enumerated.

LUNGS

"Another remarkable and almost constant lesion was the loss of epithelium from pulmonary bronchioles."
p. 10

This lesion is not mentioned in the report of Dr. Cassidy (Annex A) which described the microscopic lesions found in 10 of these dolphins. In the same report (Annex A), extensive post-mortem changes (changes occurring after the death of an animal) are described.

Since bronchiolar desquamation is a common post-mortem change in animals and humans, great care should be taken not to confuse this change, which occurs after death, with epithelial necrosis which occurs when the animal is alive.

LUNGS AND HEART

"Specifically there was pulmonary and pleural fibrosis, hepatic capsular and parenchymal fibrosis, and myocardial scarring, most common in the subendocardial region.
p.10

Cowan (1966, 1986) observed areas of fibrosis and subpleural fibrosis in the lungs of most pilot whales and dolphins that he examined and therefore considered that these lesions were frequent. If the histopathologists who examined these tissues had no previous experience of anatomical features and of common lesions found in cetaceans, a serious misinterpretation could result.

The myocardial lesions described here are frequent in cetaceans: multifocal myocardial scarring was found in 20% of normal pilot whales killed by hunters of Newfoundland[9] and subepicardial scarring was also found in 26/30 common dolphins, in 6/10 normal Pacific White-sided dolphins and in 3/6 Northern Right whale dolphins stranded on the coast of California between 1970 and 1973[10]. Therefore these myocardial lesions cannot be related with the stranding of these bottlenose dolphins since they are frequent in both normal and in diseased cetaceans.

Moreover, a layer of collagen normally thickens the endocardium of cetaceans[28]. If seen in human or in other mammalian heart, this fibroelastosis would be considered as abnormal[28].

LIVER DAMAGE

"In several animals dying late in the outbreak there was severe hepatic lipidosis, hepatocellular anisokaryosis and single-cell necrosis consistent with toxic hepatopathy." p. 10.

Not all toxins cause liver damage. Some toxins cause damage mostly to the liver while other organs are relatively spared. For some other toxins, the liver is spared while other organs are severely damaged[16]. PCBs belong to the first category, at least in chicken, mink, mice, rabbits, rats and fish[28] : toxic hepatopathy (any liver damage due to a toxin) is characteristic of PCB poisoning[11,16,17,31] while nothing is known about the target organ of brevetoxin.

All the liver changes described here such as single-cell necrosis[31], hepatic lipidosis[11,31] anisokaryosis[31] and those described in Annex A, centrilobular necrosis[31], vacuolar degeneration[11,31], bile duct proliferation[16] (4 of 10 dolphins) and periportal fibrosis[16,28] have been described in animals poisoned experimentally with PCBs. In contrast, it is not known if brevetoxin causes any damage at all to the liver (or to any other organ).

In Annex A, biliary hyperplasia was described in livers of 4/10 dolphins (livers of three dolphins were not examined). This lesion has been described in studies of PCB toxicity[16].

Hepatic lipidosis is listed in Table 3 (gross findings) but is not confirmed histologically (Table 4).

The hepatic changes described by the author on p. 10 do not always correspond with those reported in Table 4. For example, single-cell necrosis and hepatocellular anisokaryosis which are listed on p. 10 are not presented in Table 4.

LYMPHOID DEPLETION

"In many dolphins lymphoid follicles in spleen, lymph nodes, and intestine were depleted. The centers of the follicles were hyalinized, and lacked lymphocytes." p. 10

"They (the dolphins) manifested an array of chronic disorders including fibrosis of liver and lung, adhesions of abdominal and thoracic viscera, and secondary microbial infections associated with immune suppression, as evidenced by histological changes in lymph nodes." p. 17.

In the dolphins, immunosuppresssion was associated with generalized infection by a variety of opportunistic bacteria (septicemia). PCBs are potent immunosuppressors and cause lymphoid depletion, similar to what was observed in the dolphins, in most animal species[23] (Table 1 of this review). PCBs were also found in high amounts in all carcasses.

In contrast, brevetoxin is not known as an immunosuppressor. Brevetoxin has even been specifically reported as not decreasing humoral immunity in the mouse[4]. A compound assumed to be brevetoxin by the author was found in only half of the carcasses examined (8/17), and nothing is known about the significance of the levels.

BACTERIOLOGY

Bacteria found in the carcasses were various and are opportunistic; opportunistic bacteria invade hosts of which the immune system has been already weakened by other events: for instance infections by certain viruses, certain toxic compounds, stress and radiation can all cause such an event, that is, immunosuppression.

BIOTOXINS

Gymnodinium (*Ptychodiscus*) *brevis* is a dinoflagellate (protozoa), part of the phytoplankton. The lysed (broken) cells release a variety of different toxins which are neurotoxic (they impair nerve functions) and hemolytic (they break red cells). Note that although hemolysis has been reproduced in test tubes, there is no indication that any hemolytic effect causes death of fish or mice[22]. Lesions suggestive of hemolysis (hemosiderosis and extramedullary hemopoiesis) are present in only two dolphins. Unless they were observed in more animals, they should not be regarded as significant.

Unfortunately, the literature[4,5,8,22] concerned with the two major *Gymnodinium* toxins, T17 and T34, is limited to their biochemical characterization and their physiological effects. There have been no reports of lesions caused by the toxins.

It is known that the neurotoxicity of brevetoxin is due to depolarization of membranes, probably by interference with sodium channels. Very discrete lesions, if any lesion at all, are to be expected with such agents. Certainly, none of the lesions described in this report suggests a neurotoxic agent.

Although brevetoxin is a mixture of two toxins (and possibly more), the report does not mention which toxin was assayed and what were the standards used to compare peaks.

It has been reported that sluggish bottom dwellers (catfish, mullet, eels and horseshoe crabs) are affected first by Gymnodinium toxins and that finding carcasses of these animals is the first sign of an outbreak[27]. Such an event is not mentioned in the report.

ORGANOCHLORINES

Polychlorinated biphenyls (PCBs) are organochlorine compounds (OC). They are synthetic chemicals persisting in the environment, in animals, and in humans because they are protected from metabolic degradation by a ring of chlorine atoms. When ingested, they are first collected into highly perfused organs such as the liver[8]. Among other lesions, PCBs produce atrophy of lymphoid tissue (decrease in number of lymphocytes) in most animal species, thereby decreasing immune function[25]; in liver, they cause lipidosis, severe subcapsular and midzonal necrosis and in skin, they cause parakeratosis (parakeratotic hyperkeratosis).

"Three patterns were evident. 1) For PCBs, a number of dolphins showed higher concentrations in liver than in blubber, indicating that liver was not eliminating compounds at the same rate at which they were being delivered from the blubber (Fig. 3)"
p. 13

PCB concentrations in liver lipid exceeded those in blubber in 12/53 dolphins or 21% (fig. 3). The author argues that there was mobilization of lipid to explain these high levels of PCBs in the liver. If there had been mobilization of lipids, emaciation would have been noticed. In T. truncatus (bottlenose dolphins), precise measurements of weight and length determine if a dolphin is emaciated or not[23]. This information is absent from the report and thus it cannot be concluded that animals mobilized their lipid. Moreover, there are indications that dolphins did not mobilize their lipids. Table 7 shows that the lipid percentage was the same in blubber of the diseased and captive animals. Recent ingestion of high levels of PCBs is the most likely explanation for the high levels of PCBs found in the liver.

"High organochlorine levels in T.truncatus were not restricted to the stranded group; the captive had concentrations similar to those in all but the stranded mature males. The results from the beach-cast specimens obviously reflect the levels of contaminants in the nearshore environment, where the dolphins accumulate these substances. The residues occur in the

blubber of captives perhaps because they are given contaminated food, or more likely because with a steady diet, they have no need to mobilize blubber fat which could deliver the compounds to liver for excretion. Under these stable conditions, the presence of organochlorines in blubber may not pose a risk. Free-ranging animals facing intermittent food supply, or mobilizing fat during lactation, migration or times of illness, release compounds from this depot into vital perhaps more critical organs such as liver."
p. 16

A major argument used by the author for discounting any role of PCBs in the strandings is that PCB levels of captive bottlenose dolphins were comparable to levels of stranded animals. No mention of the origin of these captive animals or of the time they spent in captivity is present in the report.

This immediately raises the following questions: Were necropsies of these animals done? When were they captured, shortly before the strandings occurred? What was the cause of their death? Were there any lesions similar to those of the other dolphins, also consistent with organochlorine poisoning, or was there any disease related with immunosuppression?

It is assumed by the author that the animals mobilized lipids from their blubber and with them, PCBs. Indeed, it has been demonstrated in rats and birds that when food intake of animals contaminated with PCBs is reduced, PCBs migrate from the fat into the liver to cause severe damage[1]. However, there is no evidence of emaciation and, consequently, of mobilization of lipids in the dolphins. Blubber thickness or measurements of length and weight[23] are necessary to determine emaciation in the bottlenose dolphin: none are mentioned in the report. Some data even suggest that there was no mobilization of lipids: the lipid percentage of blubber is the same in the diseased and in the captive animals (Table 7).

After ingestion, PCBs, like most lipophilic compounds, are collected first in the liver[8,17] and this results in high liver levels. This is not considered in the report. No effort was made to determine if the dolphins ingested large amounts of PCBs even if marine sewage dumping is an important mechanism of introduction of PCBs into the environment[8]. No fish, contained in the dolphins stomach or caught offshore, were analyzed for PCBs.

"Considering the evidence that at least some dolphins were mobilizing PCBs from blubber to liver, it is conceivable that blood levels rose and were sustained long enough to exert an effect."
p. 16

At last, a possible role for PCBs is considered. This is different of what was released to the media. The role of PCBs is "conceivable" when brevetoxin is confidently assumed to play a major role in the stranding even though nothing is known about the lesions caused by this toxin. Except for the presence in 8/17 dolphins of brevetoxin, there is no evidence of brevetoxin toxicity.

"Typically affected are liver and skin, and nervous, reproductive and immune systems. Yet we cannot categorically relate any of the conditions observed in the dolphins to the known effects of these compounds (organochlorine) because of vast differences in response within and between species".
p. 16

I wish that the enthusiasm shown by the author about brevetoxin would have been tempered by the same reserve he has for PCBs especially when effects of PCBs, by contrast with brevetoxin, 1) are well known, 2) have been reproduced rather consistently for more than two decades in a variety of animals and 3) are consistent with many lesions observed in the stranded dolphins (Table 2).

It is true that there are differences between species in terms of response to PCBs but these differences are generally related with the severity of the damages caused in target organs and with the amount of PCBs necessary to cause the lesions. For instance, primary effects of PCBs in chickens, rabbits, rats and mice are limited to the liver[7]. PCBs cause skin hyperkeratosis in cows, rabbits' humans, monkeys, guinea pigs, mice and cause atrophy of lymphoid tissues in most animal species[26]. It is also important to keep in mind that differences in sensitivity of various animal species may play both ways, that is, dolphins might be very susceptible to the effects of PCBs.

"<u>The timing of the outbreak would have required that these compounds be mobilized to functionally toxic levels within a synchronized time-pulse. This is an unlikely scenario for substances which for decades have been a constant ingredient in their environment and body tissues, unless something else triggered their release by first debilitating the dolphins.</u>" p. 16-17

The author assumes that PCBs were mobilized from the blubber lipids. This is possible but there is no evidence for it. Ingestion of large amounts of PCBs by the dolphins would have also caused high PCBs level in the liver. In monkeys[2], rats[3], rabbits[20] and chickens[30], ingestion of high amounts of PCBs for few weeks result in high hepatic levels without any requirement for mobilization of lipids.

CONCLUSION

In summary, in spite of many inconsistencies and contradictions found in this report, I agree with some of the conclusions that a primary event decreasing host defenses, common to all deaths and acting on a short period of time, occurred. A toxin is a logical candidate for the etiology. However, I disagree on the relative roles played by PCBs and brevetoxin for the following reasons. Firstly, large amounts of PCBs were detected in all analyzed animals and, of the lesions produced by PCBs experimentally, many were found in the carcasses. Secondly, the lesions caused by brevetoxin are not known. Finally, brevetoxin was found in amounts of which the significance is unknown in only half of 17 carcasses and in a total of...4 fish.

Most likely, PCBs played an important role in the stranding of these animals. Levels similar to those found in these dolphins have been found to be consistently detrimental to a variety of animals.

I propose that the animals recently ingested unusually high levels of PCBs. This would explain the high levels found in the livers. This ingestion was superimposed on an already high body burden. An alternate explanation holds that the high OC levels found in the tissues of these animals, and particularly PCB levels, represent a constant threat which is fully manifested when even a low intensity stress, such as food scarcity, occurs. Then, animals mobilize their lipid and the PCBs contained in lipid are released into the blood flow.

In both cases, detrimental changes of liver, immune system and skin ensue and PCB-induced immunosuppression would cause the final demise of the dolphins. It is impossible to rule out that another toxin, such as brevetoxin, was involved but in view of the aforementioned data, PCBs certainly played an important role in the strandings.

Finally, far from being comforting, the finding of high levels of PCBs, which are toxic compounds, in randomly sampled "normal" dolphins of the North Atlantic is rather distressing.

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Table 1. Relation between levels of PCBs in tissues of animals, and lesions caused by their toxicity.

Species	SPECIMENS	Tissue levels of PCBs (ppm) associated with: Decreased humoral immunity	Lymphoid depletion
mice	liver (ww)	2.6-7.8[18]	---------
monkeys	liver (ww)	--------	0.62-12.17[2]
Dolphins	liver (ww)	10.3±13.7	
	Subcutaneous fat (lipid weight)	181.6±141.4	

ww: wet weight
lipid weight: concentration in extractable lipids

Table 2. Levels of PCBs which caused significant lesions in liver of laboratory animals

Species	SPECIMENS	PCBs concentrations	
		ppm wet weight	ppm extractable lipids
Rats	liver	16[3]	-------
Rabbits	liver	236[29]	-------
Monkeys	Mesenteric fat	5[21]	5-140[21]
Dolphins*	liver	10.3±13.7	145.7±161.6
	Subcut.fat	----------	181.6±4.4

*: final report of Dr. Geraci

ANNEX A

Interim Bacteriology Results - Dolphin Deaths Investigation - Virginia Beach, Virginia

August 29, 1987

D. R. Cassidy, National Veterinary Services Laboratories (NVSL), Coordinator

The following interim bacteriology results are reported by individual dolphins from which various tissues were submitted. The results of study of dolphin specimens submitted from the Brigantine Stranding Center were transmitted on August 29, 1987.

NVSL Accession No.	Dolphin No.	Results
40547	WAM-230	*Edwardsiella tarda* (liver, pancreas) *Streptococcus sp.** (brain, heart, lymph node, liver, kidney) *Vibrio alginolyticus* (brain, heart, lymph node, liver, kidney, pancreas) *Vibrio sp.** Possible *V. harveyi* (kidney)
40364	WAM-227	*Edwardsiella tarda* (lung, heart, lymph node) Unidentified marine *Vibrio sp.* (pancreas, kidney, liver, spleen)
40731	WAM-232	*Edwardsiella tarda* (lung, liver, spleen, kidney, heart) *Vibrio sp.** Possible *V. harveyi* (lung) *Cunninghamella sp.** (lung)
40732	CWP-263	*Streptococcus equisimilis* (spleen, liver, lung, heart, kidney) *Vibrio sp.** Possible *V. parahaemolyticus* (lung)

*determination of species and/or typing not completed at this time

UVSL Accession No.	Dolphin No.	Results
40735	CWP-264	Edwardsiella tarda - (heart, lung, kidney, liver, spleen
		Vibrio sp.* Possible V. harveyi (liver, kidney, heart)
		Vibrio sp.* Possible V. parahaemolyticus (spleen, liver, kidney, lung)
		Unidentified marine Vibrio sp.* (lung)
		Streptococcus sp.* (liver)
40788	WAN-236	Edwardsiella tarda (palate lesion)
		Escherichia coli (palate lesion)
		Pseudomonas putrefaciens (oral cavity)
		Vibrio sp.* Possible V. harveyi (palate lesion)
		Vibrio sp.* Possible V. parahaemolyticus (tongue, oral cavity, palate lesions)
41566	WAN-239	Vibrio sp.* Possible V. parahaemolyticus (lung, liver, spleen, kidney, intestine)
		All tissues were cultured anaerobically. No anaerobes were isolated.
39831	VA-206	Klebsiella pneumoniae
		Edwardsiella spp.* (spleen, abdominal fluid)

*determination of species and/or typing not completed at this time

NVSL Accession No.	Dolphin No.	Results
		Acinebacter lwoffi (urine, blubber-lesion in tail stock)
		2 *Vibrio spp.** (urine, spleen, blubber)
	VA-208	*Edwardsiella tarda* (left lung)
		Pseudomonas putrefaciens biotype 2 (abdominal fluid, left lung)
		2 *Vibrio spp.** (lung, abdominable fluid)
	VA-1	*Edwardsiella tarda*
39893	WAM-209	*Pseudomonas putrefaciens* biotype 2 (heart, blood, pancreas)
		Edwardsiella tarda - (pancreas)
		*Bacillus sp.** (heart blood, thoracic and abdominable fluid
		*Vibrio sp.** (lung, lung associated nodule, thoracic fluid)
		No growth from spleen of this animal.
	WAM-210	No growth from skin abscess.
		Pseudomonas putrefaciens biotype 2 (abdominable fluid, lung, blubber)
		*Vibrio sp.**

*determination of species and/or typing not completed at this time

NVSL Accession No.	Dolphin No.	Results
	WAM-214	Edwardsiella tarda - lung, liver, spleen
		Pseudomonas putrefaciens biotype 2
		Vibrio sp.* - (mammary fluid, abdominal fluid - no growth)
40363	WAM-226	Vibrio sp.* - (from all tissues)

/s/ L. K. Schlater Linda K. Schlater, D.V.M.
by DKC.

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*determination of species and/or typing not completed at this time.

Interim Histopathology Results - Dolphin Deaths Investigation Virginia Beach, Virginia

August 29, 1987

D. R. Cassidy, National Veterinary Services Laboratories (NVSL), Coordinator

The following interim histopathology results are reported by individual dolphins from which various tissues were submitted. The results of study of dolphin specimens submitted from the Brigantine Stranding Center were transmitted on August 29, 1987.

Dolphin No.	Case No.	Results
WAM-208	87RA500	Lung - Advanced postmortem autolysis obscures, significant morphologic detail; however, inflammatory changes indicative of subacute, multifocal, fibrinopurulent bronchopneumonia are present.
		Skeletal muscle - Marked diffuse, interstitial, fibrinopurulent, necrotizing myositis with severe diffuse necrotizing vasculitis; focal parasitism (unidentified - resembles protozoan).
No ID No.		Liver - Marked, diffuse vacuolar (fatty) degeneration.
		Lung - Focally extensive, pulmonary fibrosis and lymphocytic, interstitial pneumonia
		Lymph node - Moderate to marked multifocal, subacute, necrotizing lymphadenitis; numerous rod-shaped bacteria are seen in blood vessels, however this must be interpreted with caution in light of postmortem autolysis.
		Skin - Moderate, diffuse vacuolar degeneration of epithelial cells and epidermal cleft formation.
		Heart - Bacterial emboli are present within blood vessels. Occasionally there is invasion of the vessel wall by

Dolphin No.	Case No.	Results
		Skeletal muscle - Bacterial emboli are present within blood vessels; invasion of vessel wall by bacteria and mural necrosis are seen; moderate interstitial edema and moderate diffuse myocyte degeneration is seen.
		Intestine - Marked diffuse necrosis of Peyer's patches. Large helminth eggs (probably fluke) are seen.
		Mesenteric blood vessels - Have lesions similar to those in skeletal muscle vessels, in addition, septic thromboembolism is seen.
WAM- 206		Note: Advanced postmortem autolysis may obscure significant detail.
		Liver - Marked diffuse periportal fibrosis with bile duct hyperplasia and mild multifocal periportal lymphoid infiltration
		Spleen - Septic thromboembolism in dermal blood vessels with vascular degeneration and mural bacterial invasion, moderate epithelial degeneration; Focally extensive subacute pyogranulomatous dermatitis with dermal protozoal invasion (ciliates).
		Brain - Bacterial embolism and mural invasion of blood vessels.
		Adrenal - Moderate, acute multifocal, necropurulent adrenalitis with vascular bacterial emboli.
		Kidney - Moderate thromboembolism in blood vessels.
		Lymph node - Moderate, multifocal, necrotizing lymphadenitis with lymphoid depletion and vascular bacterial emboli.
		Lung - Focally extensive pulmonary fibrosis

Dolphin No.	Case No.	Results
WAM-207		Note: Advanced postmortem autolysis may obscure significant detail.
		Kidney - Bacterial embolism in renal blood vessels.
		Lung - Marked, multifocal pyogranulomatous pneumonia with bacterial thromboembolism and numerous fungal hyphae.
		Unidentified tubular organ - Luminal surface necrosis and inflammation (multifocal, severe)
WAM-227	87RA514	Liver - Moderate to marked periportal fibrosis with moderate biliary hyperplasia.
		Lymph node - Mild to moderate multifocal acute necrotizing lymphadenitis with lymphoid depletion.
		Spleen - Mild to moderate multifocal necrosis of lymphoid follicles; marked congestion.
		Heart - Marked subacute focally extensive necrotizing pyogranulomatous myocarditis. Moderate to many septate branching fungal hyphae are seen in lesions.
		Pancreas - Mild, multifocal lymphocytic pancreatitis with marked diffuse pancreatic fibrosis and moderate pancreatic atrophy
		Adrenal - Acute, moderate, multifocal cortical necrosis and hemorrhage.
		Lung - Focally extensive pyogranulomatous pneumonia with mycotic elements and bacteria present.
		Skin - Septic thromboembolism of dermal blood vessels with secondary ulceration and degeneration of epithelium.
WAM-232	87RA521	Cerebrum - Necrotizing vasculitis associated with numerous aggregates of bacteria within the lumina of affected vessels.

Dolphin No.	Case No.	Results
		Heart - Necrotizing vasculitis. Some vessels contain septic thromboemboli. Liver - Diffuse fatty degeneration, centrilobular hepatic necrosis; cholangitis; some bile duct hyperplasia. Lymph node - Absence of cortical lymphoid follicles, medullary edema, congestion, and focal necrosis associated with masses of bacteria. Spleen - Germinal centers in spleen are extremely cell poor. Focal necrosis in some germinal centers. Megakaryocytes are common. Bacteria numerous and are associated with necrosis. Skin - Deep ulcers with inflammation and infection penetrating underlying dermis and muscle layers. Some epithelial cells adjacent to ulcers contain eosinophilic intracyloplasmic globules resembling inclusion bodies. Lung - Hemorrhage, edema, fibrinopurulent pneumonia associated with abundant mycotic hyphae.
CJP-263	87RA522	Stomach - ulcerations Skeletal muscle - diffuse interstitial fibrinopurulent myositis (associated with bacterial colonies) muscle necrosis Lung - Localized congestion, hemorrhage, edema necrosis and fibrosis; purulent (necrotic) exudate containing myriads of mycotic hyphae Oral mucosa - ulcerated and infected. Spleen - White pulp: germinal centers cell poor; hemosiderosis, megakaryocytes common; areas of extramedullary hematopoesis.

Dolphin No.	Case No.	Results
		<u>Kidney</u> - Several foci of cortical necrosis; suppurative inflammation associated with bacteria and septic thrombi. (thromboembolic cortical nephritis)
		<u>Lymph node</u> - Lymphocyte depletion, medullary fibrosis, hemosiderosis
		<u>Thyroid</u> - Congestion/hemorrhage. <u>Liver</u> - Intense congestion with bile stasis.
CWP-264	87RA523	<u>Lung</u> - Interstitial pneumonia with mycotic pneumonia.
		<u>Skin</u> - Intraepethelial pustule formation ulceration inflammation, necrosis of underlying muscles, subcutaneous vessels contain septic thrombi.
		<u>Heart muscle</u> - Congestion and hemorrhage.
WAM-239	87RA541	<u>Spleen</u> - Moderate, acute multifocal splenic necrosis with bacterial colonization (multifocal)
		<u>Skin/tongue</u> - Septic thromboembolism of cutaneous blood vessels with invasion of vessel walls by bacteria; vascular degeneration.
		<u>Thymus</u> - Moderate, acute, multifocal, necrotizing, thymic adenitis with septic phlebothromboembolism of interlobular vessels and vascular degeneration.
		<u>Pancreas</u> - Focal, subacute to chronic, fibrosing interstitial pancreatitis with duct hyperplasia.
		<u>Liver</u> - Focally extensive portal mineralization, moderate multifocal periportal fibrosis and bile duct hyperplasia, marked diffuse vacuolar degeneration of hepatocytes.

Dolphin No.	Case No.	Results
		Lymph node - Moderate, multifocal necrotizing lymphadenopathy with vascular, bacterial embolism.
		Intestine - Severe, acute diffuse, lymphoid necrosis.
		Lung - Severe, diffuse subacute to chronic pneumonitis with nematodiasis. Severe, acute to subacute fibrinohemorrhagic to fibrinopurulent bronchopneumonia with fungal hyphae.
		Brain - Moderate, diffuse, vascular degeneration with mild vasculitis, bacterial embolism, and mural invasion by bacteria.
WAM-209	87RA501A	Note: Tissues are in state of moderate to severe postmortem autolysis
		Kidney - Bacterial embolism in renal blood vessels. Tubular epithelial degeneration (slight reminiscence of intranuclear inclusions in tubular epithelial cells.
		Lymph node and Spleen - Possible necrosis but too much autolysis.
		Liver - Severe diffuse vacuolar (fatty) degeneration of hepatocytes.
		Skin - Bacterial thromboembolism in dermal vessels with focally extensive epithelial degeneration, erosion and ulceration (epithelial degeneration characterized by vacuolation and formation of pink cytoplasmic globules within the basal epithelial cells). Pseudoepitheliomatous hyperplasia

Dolphin No.	Case No.	Results
		Brain - Bacterial emboli in blood vessels with vascular degeneration.
		Adrenal - Mild to moderate multifocal cortical necrosis with bacterial embolism.
		Heart - Bacterial embolism with vascular degeneration.

/s/ A. J. Davis

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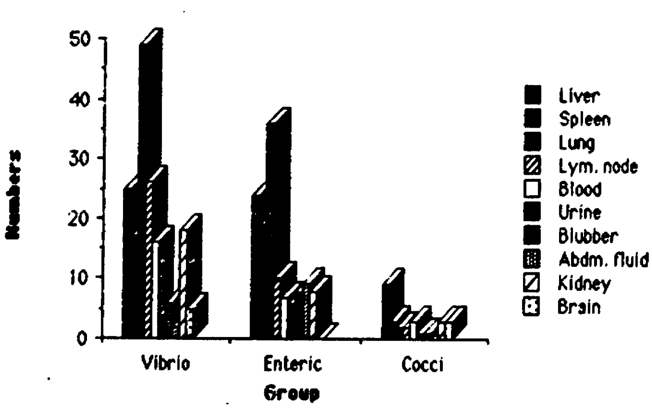
(Chart accompanying a statement of Harry L. Smith.)

Groups	Liver	Spleen	Lung	Lymph	Blood	Urine	Blubber	Ab flu	Kidney	Brain	Total
Vibrio	25	17	49	26	16	4	6	2	18	5	168
Enterics	24	19	36	10	7	6	8	9	8	0	127
Cocci	9	3	2	2	3	1	1	0	3	3	27
Total	58	39	87	38	26	11	15	11	29	8	322



PLEASE NOTE: General discussion of Report's findings begins on Page 14 (Tabbed).

CLINICAL INVESTIGATION OF THE 1987-88 MASS MORTALITY OF BOTTLENOSE DOLPHINS ALONG THE U.S. CENTRAL AND SOUTH ATLANTIC COAST

final report to

National Marine Fisheries Service
and
U.S. Navy, Office of Naval Research
and
Marine Mammal Commission
April, 1989

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INTRODUCTION

From early June, 1987, until March, 1988, unprecedented numbers of bottlenose dolphins, *Tursiops truncatus*, washed ashore along the Atlantic coast from New Jersey to Florida. Details of the initial response to the event, subsequent organization of a multi-disciplinary team of investigators, and scope of the analyses were provided in an unpublished Interim Report submitted to the U.S. Marine Mammal Commission in May 1988. An account of the extent and impact of the mortality has been prepared by Scott *et al*. (1988).

The event was unparalled, and therefore demanded a comprehensive investigation of proximate and contributing factors. Routine laboratory protocols were modified to meet rigorous research standards. Contributing laboratories with expertise in pathology, biochemistry, microbiology, virology, contaminants, and biotoxins performed analyses on coded samples from the dolphins. Specimens for contaminant and biotoxin analysis were mixed with controls from unrelated *Tursiops* and four other cetacean species. At the termination of each study, data were transferred to our laboratory at the University of Guelph, and integrated with identifying information.

This report describes how the investigative process evolved, and the evidence implicating a biological toxin as the proximate cause. The dolphins apparently were poisoned by brevetoxin, a neurotoxin produced by the dinoflagellate *Ptychodiscus brevis*, Florida's red tide organism. The dolphins were eventually infected with a host of bacterial and viral pathogens which produced an array of beguiling clinical signs.

MATERIALS AND METHODS

Specimen Collection

Over 740 bottlenose dolphins stranded along the Atlantic coast during the 11-month period beginning June, 1987 (Scott *et al*. 1988). Data or specimens from 347 of these were available for analysis by the investigating team. Studies on pathology, virology, microbiology, and chemical and biological toxicology were carried out only on freshly dead animals (Table 1).

To examine and obtain blood samples from live animals, four bottlenose dolphins were captured just offshore along Virginia Beach on August 16, and nineteen more between October 6 and 9, 1987. Blood samples were analyzed for hematology and serum chemical constituents including electrolytes, metabolites, enzymes, proteins and protein electrophoretic patterns, thyroid and adrenocortical hormones, and viral antibodies.

Pathology

Tissues for pathologic examination were fixed in 10% buffered formalin. Samples were processed through alcohol and xylene and embedded in paraffin blocks. Sections 5 µm thick were stained with hematoxylin and eosin, Masson's trichrome, Brown and Brenn, methenamine silver, Von Kossa, or periodic acid Schiff.

Selected samples of lung tissue were processed for electron microscopy. They were transferred to glutaraldehyde, post-fixed in osmium tetroxide, dehydrated in acetone and embedded in epon. Thick sections, 0.5-1 µm, were cut on a Reichert-Jung Ultracut E ultramicrotome[1], and stained with methylene blue. Ultra-thin sections of subsamples were stained with uranyl acetate and lead citrate and examined on a Hitachi HS-9 electron microscope.

For energy dispersive x-ray analysis, samples of lung were processed without osmium tetroxide. Ultra-thin (90 nm) sections were collected on nickel grids, and examined in a low-background beryllium holder. Mineralized deposits were characterized for elemental composition using a Phillips EM 400T/STEM/TN (Tracor Northern) 5500 Series 1 Energy-dispersive X-ray Analyzer. Sections were bombarded with electrons for 100 live seconds at an accelerating voltage of 100 kV with an electron probe size of 400 nm. Beam current conditions were standardized for each analysis. Deposits were probed at three sites progressing from the core to the outer edge; an adjacent area of lung was analyzed for background elemental composition.

Virology

Specimens were submitted to the Eastern Virginia Medical School (EVMS), the USDA-NVSL at Ames, IA, and the National Institute of Health (NIH). At EVMS, under the direction of Dr. K. Somers, tissues and lesions from 12 dolphins were examined for the presence of viruses by electron microscopy, immunofluorescence, and cytopathic effects in tissue culture. Monoclonal antibodies specific for influenza A and B, parainfluenza 1 and 3, varicella-zoster virus, herpes simplex 1 and 2, and adenovirus were used to test for the presence of viral antigens. Tissue extracts were inoculated into cell cultures of monkey kidney, human skin, human carcinoma (Hep-2, A549), and mink lung.

At the USDA-NVLS, under the direction of Dr. L. Peterson and Mr. G. Gustafson, virus isolation was attempted on 54 tissue specimens from 29 dolphins. A 10 percent tissue suspension was prepared and inoculated into embryonating chicken eggs (ECE) and cell cultures (CC). The number of specimens inoculated into ECE was as follows: yolk sac route - 34; allantoic route - 27; chorioallantoic membrane route - 27. The number of specimens inoculated onto each cell line was: Vero-M - 49; McCoy -34; Madin Darby canine kidney - 9; baby hamster kidney - 10; bovine turbinate - 23; dolphin kidney - 3; dolphin skin - 2. The following is the number of specimens inoculated onto primary cell cultures: chick embryo

[1] The use of brand names is not intended to indicate or imply an endorsement for the named equipment of product.

kidney - 17, rhesus monkey kidney - 9, swine kidney - 23, and swine buffy coat - 5. Each specimen was passed at least two times in cell culture and/or ECE. The ECE were observed for embryo death and the allatonic fluid was tested for hemagglutonating viruses, influenza and parainfluenza viruses. The cell cultures were observed for cytopathic effect and examined by electron microscopy for viral particles. Thirty-five of the original tissues submitted to the USDA-NVSL were also examined by electron microscopy for viral particles.

Responding to public concern that the dolphins might have been infected with retroviruses such as that responsible for AIDS in humans, Dr. R. Benveniste of the National Cancer Institute, NIH, examined 17 blood samples taken from live dolphins. Peripheral blood lymphocytes were co-cultivated with normal human peripheral blood lymphocytes, human lymphocyte lines HuT78 and MOLT3, human monolayer cell line A549, and canine monolayer cell line FCf2TH. These cell lines support the growth of almost all known mammalian retroviruses, including human immunosuppression virus. Table 2 summarizes the results of this and other efforts to isolate and identify viral agents in dolphin tissues.

Bacteriology

Bacteriological studies were carried out at USDA-NVSL, the Virginia Beach General Hospital, and the Center for Disease Control (CDC), Atlanta, GA. Tissues and swabs were submitted on wet ice. Swabs for aerobic culture were submitted in Cary-Blair transport medium (catalog no. 06-0452, Remel, Lenexa, KS). Swabs for anaerobic culture were submitted in anaerobic specimen collectión kits (catalog no. 3650, Becton Dickinson, Rutherford, NJ). All specimens were processed as soon as possible after arrival at the laboratory.

Tissue specimens and swabs submitted for aerobic culture were inoculated onto marine agar (Difco, Detroit, MI), MacConkey agar (Difco), and heart infusion agar (Difco) supplemented with 5% defibrinated bovine blood. These media were incubated at 37°C for 24 hours and at room temperature for an additional 48 hours. Swabs for anaerobic culture were inoculated onto anaerobic blood agar (Dowell and Hawkins 1981), and incubated at 35°C in an anaerobic glove box (Forma Scientific, Marietta, OH). Plates were examined for anaerobes after 24 hours and 48 hours incubation. The freshest tissue specimens were also inoculated onto charcoal yeast extract agar (CYE, Remel) in an attempt to isolate fastidious organisms which might not grow on blood agar. The CYE plates were incubated at 37°C in a CO_2 incubator (Model 5200, National Appliance Co., Portland, OR) and examined daily for 1 week.

The methods used for the biochemical characterization of isolates were essentially those of Edwards and Ewing (1986) and Clark _et al_. (1984). For characterization of _Vibrio_ isolates, the following media were supplemented with sodium chloride (3% final concentration): indole, methyl red, Voges-Proskauer, malonate, nitrate, gelatine, and decarboxylases (Moeller). Heart infusion broth containing 1% (wt/vol) carbohydrate, 3% (wt/vol) sodium chloride, and 1.0% (vol/vol) Andrade's indicator was used for fermentation tests. For salt tolerance tests, nutrient broth containing increasing concentrations (0%, 3%, 6%, 8% 10%) of sodium chloride was used. All biochemical tests for Vibrio spp. were incubated at 25°C. Biochemical tests used for characterization of organisms other than Vibrio spp. were incubated at 37°C.

Serology

At the USDA-NVSL, serum samples from live-captured dolphins were tested for antibody as follows: bovine leukosis, equine infectious anemia, ovine progressive pneumonia and bluetongue by immunodiffusion (ID); contagious ecthyma, chlamydia and Coxiella burnetii by complement fixation (CF); equine rhinopneumonitis, equine coital exanthema and equine herpes-2 by serum neutralization (SN); vesicular stomatitis by CF and SN; and African swine fever by enzyme-linked immunoassay test.

At the OMAF-VLS laboratory, Dr. S. Carman conducted standard virus neutralization microtiter assays to determine titers for serum antibodies to canine distemper virus (CDV) in samples collected from 13 dolphins captured alive off Virginia Beach in October, 1987. Two-fold serial dilutions of heat-inactivated (30 min at 56°C) test sera were mixed with equal volumes of Onderstepoort strain of CDV virus (originally obtained from R.C. Povey, OVC), containing 100 $CCID_{50}$. The mixtures were incubated at 4°C for 1 h, after which Vero cells were added. Plates were incubated for 4-5 d at 37°C in a humidified CO_2 incubator. Sera were tested in duplicate, along with known positive and negative canine sera as controls. The titer was determined as the dilution of serum that completely inhibited virus replication in 50% of the wells, or the 50% end-point was extrapolated.

Toxicology - Chlorinated Hydrocarbons

Analyses were performed at the USDA-NVSL, in the laboratory of Dr. H. Nelson and F. Ross. To reduce contamination, specimens of liver, blubber, brain and kidney were removed as soon as possible after the carcass was opened. Tissues were wrapped in aluminum foil, placed in plastic bags, frozen at -20°C, and shipped to the laboratory where they were stored frozen for up to 1 year. Included for comparative purposes were specimens of stranded pilot whales, Globicephala melaena (4 mature F, 3 immature F, 1 mature M, 1 immature M, 2?), harbor porpoises, Phocoena phocoena (1 mF, 3 iF, 4?), humpback whales, Megaptera novaeangliae (2 mF, 3 iF, 1 mM, 1 iM, 1?) and three captive Tursiops (2F, 1M). The tissues were collected and stored as described above, except that they were placed into plastic bags, without aluminum foil, and storage times ranged from 2 to 10 years.

Five gram samples of blubber and melon (when available, cortex from melon was also taken) were shaved into thin (1 to 2 mm) slices, diced, placed into a tared 100 mL beaker and weighed. Liquid nitrogen (25 mL) was poured over the material to disrupt the cells. Once the liquid nitrogen evaporated and the beakers had returned to room temperature, 10 g of Na_2SO_4 was added as an abrasive to facilitate maceration and to scavenge moisture. A robust glass rod was used to press and macerate the material against the bottom of the beaker. One hundred mL of methylene chloride ($MeCl_2$) was added, and the covered beaker gently agitated on a platform shaker (100 rpm) at room temperature for 24 hours.

The $MeCl_2$ was then filtered through filter paper into a tared evaporating flask. The beaker was rinsed twice, each time with 50 mL of $MeCl_2$, and the rinseates were filtered into the same flask, which was then placed in a rotary evaporator with a water bath temperature of 44 to 47°C to remove the $MeCl_2$. The residue was weighed to determine the amount of lipid.

Liver samples were homogenized in a Waring blender, and a 10 g sub-sample was combined with 20 g of Na_2SO_4. The slurry was mixed with a wooden stirrer, weighed, and dried at 80°C for 24 hours; moisture content was determined by reweighing the dried preparation. One hundred mL of $MeCl_2$ was then added, and after gentle agitation, filtered through paper into a tared evaporating flask. The beaker was rinsed twice with 50 mL $MeCl_2$ and the rinseates filtered into the same flask. The flasks were placed on a rotary evaporator to remove the $MeCl_2$, then weighed to determine the amount of lipid.

The lipid extract from each liver, blubber, and melon sample was dissolved in 10 mL of equal volumes of $MeCl_2$ and cyclohexane. For blubber and melon samples with lipid yield greater than 2 g, 1 g of lipid was weighed into a 15 mL glass tube and used for subsequent analyses. Five mL of the solution was loaded onto a gel permeation chromatograph (GPC) (AutoPrep[R], GPC Analytical Biochemistry Laboratory, Columbia, MO). The GPC was equipped with a 60 X 2.5 cm i.d. chromatographic column packed with a 60 g BioBeads (BioRad[R], Cambridge, MA) SX-3 resin in a 48 cm bed. MeCl2:cyclohexane (1:1) was pumped at 5 mL/min to elute the column. Samples were fractionated according to American Organization of Analytical Chemists (AOAC) Official Methods of Analyses (14 ed., 1984, Section 29.037-29.043). One hundred fifty mL of eluate containing chlorinated hydrocarbons was collected into an evaporating flask, and the solvent removed. Ten mL of petroleum ether (PE) was added in 3 aliquots to the residue and the solution transferred to a column containing 20 g Florisil[R] (60/100 PR grade provided by U.S. Food and Drug Administration, Minneapolis, MN). Two fractions were collected (fraction A, 200 mL PE; fraction B, 200 mL 50/50/0.5, PE/MeCl2/acetonitrile, V/V/V) in flasks according to AOAC Methodology Section 29.015, and the solvent removed as described. The residue in each fraction was redissolved with three rinses totalling 8 mL of equal parts PE and acetone, and transferred to a 15 mL capped tube. The level was then adjusted to a final volume of 10 mL, after aldrin was added as the internal standard. Fractions A and B were then subjected to gas chromatographic analysis.

Polychlorinated biphenyls (PCB, as Aroclor[R] 1260) were quantified from fraction A on a Perkin Elmer 8500 Gas Chromatograph equipped with a Ni63 electron capture detector, a Perkin Elmer As 8300 Autosampler (Norwalk, CT), and a 15 m x 0.25 mm DB-5 (J and W Scientific, Folsom, CA) fused silica capillary column with splitless injection. The carrier gas, 5% methane in argon, was delivered at a flow rate of 1.0 mL/minute. Separation was obtained using a temperature program from 150 to 240°C at 2°C/minute with a 5.0 minute post-injection hold at 150°C. The PCB's eluted in a time window from 21 to 52 minutes, and were identified by GC retention times using congener standards (Muir et al. 1988) obtained from the National Research Council of Canada (Marine Analytical Standards Program, Halifax, N.S), and also with gas chromatography/mass spectrometry (GC/MS). Quantitation was done by summation of all PCB chromatographic peaks identified by comparison with an Aroclor® 1260 standard obtained from the U.S. Environmental Protection Agency (EPA) (Las Vegas, NV).

Chlorinated pesticides (DDT group and trans-nonachlor) were quantified on a Perkin Elmer Sigma 1 Gas Chromatograph equipped with a Ni63 electron capture detector, a Perkin Elmer As 300 Autosampler, and a 2 m x 2 mm i.d. glass column packed with 1.5% SP-2250/1.95% SP-2401 on 100/120 Supelcoport (Supelco, Inc., Bellefonte, PA). The carrier gas was 5% methane in argon used at a flow rate of 40 mL/minute and the oven temperature was 200°C isothermal. Pesticides were quantified by comparing with EPA authentic standards. Trans-nonachlor and p,p'DDE were measured in fractions A and B, then totalled.

The identities of PCBs and pesticides were confirmed in selected liver, blubber, and melon samples on a Finnigan/MAT TSQ 70 Tandem Mass Spectrometer equipped with a 5 m x 0.25 mm DB-1 (J and W Scientific, Folsom, CA) capillary column with splitless injection operated at a flow rate of 1 mL/minute helium. Fragmentation was by electron impact or methane chemical ionization. Spectra were identified by comparing them to the NBS-NIH Mass Spectral Library and to standards. The lower limit of sensitivity was estimated to be 0.1 ppm for chlorinated hydrocarbons and 1.0 ppm for PCB. Values below these limits were considered zero in statistical computations.

Liver and blubber samples were processed in batches representing ten animals. A positive control was prepared for each batch from pesticide-free bovine fat with known amounts of chlorinated hydrocarbons added. Mean percent recovery and reproducibility for these spiked samples (X% ± standard deviation) was: p,p'DDE (liver) 90 ± 9; p,p'DDE (blubber) 85 ± 8; PCB (liver) 92 ± 7; PCB blubber) 87 ± 9; nonachlor (liver) 89 ± 8; nonachlor (blubber) 80 ± 7. No adjustments in results were made on the basis of these recoveries. True reproducibility measurements were obtained from analyses of five pairs each of blind duplicate liver and blubber samples, spaced throughout the course of the analyses. The average percent difference between the duplicate values was 14% for PCB, 12% for DDE, and 10% for t-nonachlor. The precision of the blubber lipid extraction was determined by conducting ten separate determinations on a randomly selected blubber sample. Average lipid yield was 67.5%, with a standard deviation of 1.4%.

Sub-samples of dolphin tissues or extracted lipid were sent to the laboratories of the Canadian Department of Fisheries and Oceans, Bedford Institute of Oceanography (BIO), Halifax, N.S. and Massachusetts Department of Public Health, Boston, MA for independent verification of DDE, PCB (total and congeners), and extractable lipid. Blubber lipid results from BIO were systematically lower for samples above 50%, and equivalent for those below that level. Liver lipid results were in excellent agreement, with an overall average percent difference of 15% for 10 samples. Reproducibility on 17 of 20 blubber samples for PCB and DDE was 15% and 13%, respectively, with three outlier results attributable to different blubber lipid yield. Liver PCB and DDE reproducibility was 20% and 13%, respectively, on 10 samples.

Toxicology - Elemental Analysis

Liver from the dolphins was collected and stored in the same manner as described for hydrocarbons. One gram of blended liver was weighed into a 15 mL teflon screw-top vial (AOAC Official Methods of Analysis, 14 ed. 1984, Section 49.A01 to 49.A05). Five mL of nitric acid (Mallinckrodt AR Select[R], Mallinckrodt, St. Louis, MO) was added, and the capped vial was positioned in a shallow glass dish containing 100 mL H_2O and placed into a 400 watt microwave oven, where the sample was digested for 2 to 3 minutes. After cooling, the material was filtered through filter paper into a 25 mL acid-rinsed volumetric flask; scandium was added as the internal standard.

Liver samples were processed in batches of ten. National Bureau of Standards reference materials (1577a and 1566) and normal bovine liver with lead, selenium, and mercury added were run with each batch. Quantification of the elements was carried out on a Perkin Elmer Model 6500 Inductively Coupled Argon Plasma Emission Spectrometer (ICP) equipped with a Czerny-Turner 408 mm focal length monochromator with holographic grating (UV, 2880 lines/mm and visible, 1440 lines/mm). Individual emission lines were as follows:

Element	Wavelength (nm)	Element	Wavelength (nm)
Copper	324.754	Cadmium	214.438
Zinc	213.856	Lead	220.353
Selenium	203.985	Mercury	194.227

Measurements were made using the sequential or graphic mode. Individual elements were quantified against primary standards prepared according to recommended procedures (Perkin-Elmer Procedure Manual) or obtained from a commercial source (Fisher Scientific Co., Pittsburgh, PA). Mercury standards were digested the same as the samples; all other standards were diluted from stock.

Toxicology - Biotoxins

Liver samples were taken from 18 of the freshest dolphins, selected to represent three arbitrary phases during the event: early (5 between August 8-26, 1987), middle (5 between September 18 - October 8, 1987), and late (8 from December 13 - February 19, 1988). These were tested in the laboratories of the Massachusetts Department of Public Health, for the presence of saxitoxin (STX), a water-soluble neurotoxin produced by a marine dinoflagellate which is responsible for paralytic shellfish poisoning (PSP). Three that died during capture in October, 1987, were tested as controls. Each test involves intraperitoneal inoculation of tissue extract into mice, as a bio-assay screening procedure. Samples found to be positive on bioassay are then processed by extraction and purification, and active compounds identified and quantified using high pressure liquid chromatography (HPLC).

Sixteen of the liver samples from the beached dolphins that were tested for saxitoxin, and one additional dolphin liver specimen, were also analyzed for brevetoxin produced by Ptychodiscus brevis, the dinoflagellate responsible for the "red tide" phenomenon. Controls included the three dolphins that died during capture, and 14 additional bottlenose dolphin samples - 6 that stranded along the mid-Atlantic coast between August and November, 1988; 3 from the Texas coast (one in Feb, 1987, two in March, 1988); 1 from Cape Cod, MA, (1983); and 4 captives (3 adults, 1 calf). Analyses were carried out in the laboratory of Dr. D. Baden, University of Miami. Samples sent to the laboratory were identified by code number, and their identity revealed to the laboratory only after test results were made available to the principal investigator.

At the time of the outbreak, the possibility of biotoxin poisoning was considered, and in August, 1987, we obtained bluefish, Pomatomus saltatrix, Atlantic croaker, Micropogon undulatus, spot, Leiostomus xanthurus, and red drum, Sciaenops ocellata, from the onshore weir fishery off Virginia Beach. These were tested for PbTx. We also analyzed menhaden, Brevoortia sp., and weakfish, Cynoscion regalis, taken from the stomach of a dolphin (KDL644) stranded south of Cape Canaveral on January 13, 1988, for PbTx. This was the only suitable sample of stomach contents available in the entire collection. After preliminary results showed the presence of PbTx in some dolphin samples, we obtained 2 silver seatrout, C. nothus, 3 Spanish mackerel, Scomberomorus maculatus, and 3 menhaden, Brevoortia smithii, caught off Vero Beach, FL, by the Florida Dept. of Natural Resources in late February, 1988. Viscera were analyzed from fish individually or as a pooled sample; selected specimens of flesh were also tested using the same protocol and criteria as for the dolphin liver samples.

Dolphin liver specimens (35-275 g) were received frozen. Each sample was homogenized, then dehydrated by steeping in 2 volumes of anhydrous acetone for 10 hours, followed by vacuum filtration on a Buchner filter using coarse-grade ashless filter paper. Dehydrated samples were homogenized twice in chloroform solvent, and the solvent was removed by filtration. The acetone and chloroform filtrates were combined, discarding the solid residue. Each filtrate was flash-evaporated, the residues were each resuspended in 20-25 mL 90% aqueous methanol and were solvent-partitioned twice with equal volumes of light petroleum. Methanol fractions were retained, adsorbed to 15-30 g dry silica gel, dried, and packed into flash chromatography columns over 100 g dry silica gel. The dry columns were eluted with 2 column volumes of anhydrous acetone, and the eluates were reduced in volume to 0.5-1 mL. Samples were rechromatographed as described above, using 50-100 mL chloroform:methanol:acetic acid (100:10:1).

All eluted solvent was flash-evaporated, each residue was applied to a 20 x 20 cm 1000 μm preparative silica gel thin layer chromatography plate, and plates were developed in acetone/light petroleum (30/70). One-cm-wide fractions (5%) of each developed thin-layer plate were scraped and bioassayed using mosquito fish, Gambusia affinis. Fractions which were lethal as determined after 48 hours were scraped, eluted with acetone, and rechromatographed on 10 x 20 cm 500 μm preparative silica gel thin-layer chromatography plates using ethyl acetate/light petroleum (70/30) as solvent. Fractions of developed plates were bioassayed as described above, and active fractions were eluted with acetone. Eluted fractions were dried under a stream of nitrogen, redissolved in 250 μL HPLC grade methanol, and were filtered using a 0.2 μm nylon filter. Samples were subjected to HPLC using a C-18 reverse phase column and 85% aqueous methanol as mobile phase. Detection was by ultraviolet absorbance at 215 nm. Concentrations and identity of individual brevetoxins were determined by peak height, retention time, and comigration using brevetoxin standards prepared in the laboratory.

Results obtained using this protocol are generally adequate to confirm the presence of PbTx. Nevertheless, as an additional step, Fourier transform infrared transmission spectrometry was performed using a Mattson Instruments Cygnus 100 FTIR equipped with a Unix Starlab 2000 database and laser internal wave number standard. Extracts from one of the PbTx-contaminated livers were prepared in KBr pellets, and the spectra obtained were compared to those of authentic PbTx by computer-averaging of 32 sequential scans of each sample. Spectra were overlaid by computer and similarities were documented in the fingerprint region of each spectrum (1900-400 cm^{-1}).

RESULTS

Pathology

Necropsy and histologic findings in organ systems found to have the most consistent pathologic disorders are summarized on Tables 3 and 4. The study required numerous observers over a broad geographic area. Inconsistencies in reported findings were therefore inevitable. Despite the limitation, trends were noted in the condition of the stranded dolphins. Those that came ashore in August and early September 1987 had a range of skin lesions. Small blisters and pock-like craters were common over the head region, particularly around the lips and snout, and in the soft tissues of the mouth. The dorsal fin, flippers, and tail flukes were also affected to some extent. Rarely, the entire surface of the body was covered with round raised pox-like lesions measuring up to 1 cm in diameter. Histologically, the lesions consisted of vacuolation and swelling of epidermal cell cytoplasm with no involvement of the dermis. A viral infection was suspected, and though inclusion-like structures were occasionally noted, they contained no convincing evidence of virus particles. Results of viral isolation from representative lesions are reported below (see Virology).

A second type of skin lesion noted was the sloughing of large areas of skin, exposing underlying intensely reddened dermis. In some cases, large blisters formed and coalesced into broad sheets of epidermis floating on a fluid-filled bed. The epidermis could be peeled back as easily as a covering of cellophane. This condition could be distinguished, both by character and cause, from the pox-like lesions. These lesions were associated with thrombosis of dermal vessels, presumably caused by bacteria, fungi, or protozoa. This condition was one manifestation of systemic bacterial invasion which seems to have been the ultimate cause of death of many of the dolphins during the hot summer months. As time progressed, fewer of these lesions were noted, whereas the pox-like condition on the lips and snout was still evident in dolphins recovered in late February 1988.

Other findings in the dolphins were also related to septicemia, and particularly to the effects on blood vessels which had been injured by bacteria. The vessel walls became fragile and necrotic, and were unable to contain blood. Plasma leaked into tissue spaces causing edema in many of the organs, and accumulation of massive quantities of blood-tinged fluid in the thoracic and abdominal cavities. Affected organs underwent necrosis as a combined effect of impaired circulation and bacterial toxins. In some cases the animals appeared to have died during the acute phase of bacterial infection. Others, less severely affected, had a more protracted illness which terminated in pneumonia, cerebral hemorrhage, secondary invasion by fungal organisms, and vascular collapse or shock.

Chronic lesions were present in some of the first animals examined in early August 1987. These were typically found in the lung, liver, pancreas, and heart, and were characterised by fibrosis. Specifically there was pulmonary and pleural fibrosis, hepatic capsular and parenchymal fibrosis, and myocardial scarring, most common in the subendocardial region. Pancreatic fibrosis grossly typical of chronic parasitic infestation was also present. Fibrosis in the lung was most severe sub-pleurally and much of the "pleural" thickening was actually due to this lesion. In the few animals in which the trachea was examined histologically, chronic tracheitis was consistently present.

Another remarkable and almost constant lesion was the loss of epithelium from pulmonary bronchioles. The walls of affected airways were lined by fibrous tissue in which mineralized debris was embedded, while the few remaining epithelial cells were stretched to cover the ulcerated surface. The mineralized structures, which measured 22-26 μm in diameter, were formed of concentric rings, with mineralization most apparent in the core. Electron-dispersive analysis revealed that calcium and phosphorus were the principal elements in these structures. Their concentrations decreased progressively towards the edges of the structures, and were undetectable in adjacent lung tissue.

In liver there was thickening of the capsule and fibrosis of parenchyma especially around portal triads and under the capsule. Some of this fibrosis was associated with parasitic infestation but elsewhere the fibrosis was typical of post-necrotic scarring. In several animals dying late in the outbreak there was severe hepatic lipidosis, hepatocellular anisokaryosis and single-cell necrosis consistent with toxic hepatopathy.

In many dolphins lymphoid follicles in spleen, lymph nodes, and intestine were depleted. The centers of the follicles were hyalinized, and lacked lymphocytes.

Bacteriology

A wide variety of bacteria was recovered from stranded dolphins (Table 5). The organisms include members of the genera _Edwardsiella_, _Streptococcus_, _Vibrio_, _Pseudomonas_, _Klebsiella_, _Acinetobacter_, _Bacillus_, _Staphylococcus_, and others. There was no particular pattern to their distribution within an animal. Members of the _Vibrio_ group predominated, representing 52% of the total isolates. All tests for _Chlamydia_ were negative.

Virology

Tissue specimens and lesions from dolphins were evaluated for the presence of viruses by electron microscopy, immunofluorescence, and cytopathic effects in tissue culture. No virus particles were observed in direct examination by electron microscopy, nor were antigens detected to influenza A and B, parainfluenza 1 and 3, varicella-zoster virus, herpes simplex 1 and 2, and adenovirus. All samples were negative for bovine leukosis, bluetongue, contagious ecthyma, equine infectious anemia, equine rhinopneumonitis, vesicular stomatitis, and ovine progressive pneumonia. There was no evidence of retrovirus infection.

Papovavirus was detected in 4 of 12 dolphins, on the basis of electron microscopic examination and cytopathic effects (CPE) in primary monkey kidney cell cultures inoculated with tissue extracts. The same extracts had no effect on human skin cell cultures, human carcinoma cell lines, or mink lung cell cultures. The virus was immunologically related to simian virus 40 (SV-40) as demonstrated by immunofluorescence with antiserum specific for the VP 1 capsid antigen of SV-40. Uninfected monkey kidney cells were negative for virus particles by EM and SV-40 capsid antigens by immunofluorescence. Herpes-like particles were also isolated from a mouth lesion from one of these dolphins. At the EVMS, Dr. Somers isolated a virus related to the reovirus family, from the palate ulcer of dolphin WAM-253. The virus has a restricted host range and induces cytopathic effects in dolphin kidney cell cultures (CCL 78) (American Type Culture Collection, Rockville, MD), but fails to cause cytopathic effects in human fibroblast or epithelial cells, mink lung cells, monkey kidney cells, and rabbit kidney cells; uninfected dolphin cell cultures show no evidence of the virus. The CPE occurred after a 2-3 day latent period, were reproducible, and consisted of cell clumping, apparent cell-to-cell fusion, ballooning degeneration, and lysis. Ballooning cells extruded transparent cytoplasmic extensions from the surface membrane. Electron microscopy of infected dolphin cells (CCL 79) (American Type Culture Collection, Rockville, MD) revealed the presence of virus particles 75-80 nm in diameter which accumulated in the cytoplasm. There were no intranuclear forms. The size, shape and localization of the virus was consistent with a reovirus identity. Reovirus-like particles were isolated from a palate ulcer from a fifth dolphin. The isolate induced reproducible CPE in dolphin kidney cell cultures. All three isolates are being further characterized.

Serology

Canine distemper virus-neutralization assays on serum from live-captured dolphins showed inhibition of virus in 6 of 13 samples. Titers greater than 1:2 suggest that CDV antibody was present. One dolphin had a titer of 1:128, two of 1:24, two of 1:12, and one of 1:6. There was no apparent bias in sex or age of the positive dolphins.

Biotoxins

There was no evidence of saxitoxin on preliminary mouse bioassay of dolphin liver samples. No further analyses were performed.

The results of brevetoxin analyses are presented in Table 6. The analysis consists of three purification steps, each followed by a fish bioassay. A negative result at any stage terminated the test. Only those samples positive in the third bioassay were subjected to HPLC. Diagnosis was based on detecting a specific HPLC peak which co-migrated with the brevetoxin standard. Fourier transform infra-red transmission spectrometry performed on the extract from dolphin WAM 280 provided unequivocal evidence that the active component was PbTx-2. Comparison of the generated wave numbers revealed characteristic absorption in the fingerprint region for both samples at 3435-3441, 2940-2941, 2851-2874, 1638, and 1056 cm^{-1}.

Using these criteria, eight of 17 stranded dolphins collected during the event tested positive for the neurotoxin; two of six collected near Virginia Beach in July and August, 1987; three of five in the same area between September 18 and October 8, 1987; and three of six along northern Florida in January and February, 1988 (Table 6). There was no apparent correlation between the concentration of the toxin and the chronology or location of stranding. No PbTx-2 was demonstrated in any other dolphin liver sample, including the three animals that died during capture in October, 1987.

Brevetoxin was found in the viscera but not in the flesh of menhaden taken from the stomach of dolphin KDL644; no toxin was detected in weakfish also taken from the same animal, nor from the liver of that dolphin. Of the seven species of fresh-caught fish tested, only the viscera of menhaden landed on February 20 and 28, 1988, contained detectable brevetoxin, at levels representing 200 µg per fish.

Toxicology - Organochlorines

Results of organochlorine analyses and lipid recovery in blubber and liver are expressed on a lipid weight basis, and are shown on Tables 7 and 8. The findings for liver are also expressed on the basis of wet weight (Table 9). Three major groups of organochlorine contaminants were detected: DDTs, chlordanes and PCBs. The DDT fractions included p,p'DDE, o,p'DDE, p,p'DDD, o,p'DDD, p,p'DDT, and o,p'DDT. This group is represented by p,p'DDE. Chlordane components included trans-nonachlor (t-nonachlor), cis-nonachlor, cis-chlordane, trans-chlordane, heptachlor epoxide, oxychlordane, and heptachlor. T-nonachlor was the major component and was selected to represent this group. The chromatographic profile of the PCBs was most like that of Aroclor ® 1260, and consequently is expressed as such. In the following discussion, liver and blubber concentrations are expressed on the basis of lipid weight unless otherwise stated.

The majority of liver samples contained less than 10% extractable lipid (Fig. 1). The few samples that exceeded that value ranged up to 41%; all animals with greater than 15% extractable lipid were immature. The majority of blubber samples contained more than 50% extractable lipid (Fig. 1); values averaged 10% higher in immature males and females than in mature animals (Table 7). Data from the three dolphins with extractable blubber lipid less than 10% were atypical, and were excluded from the calculations of mean values.

Average concentrations of organochlorines in blubber were higher in immature than in mature females, and showed the opposite pattern in males. Statistically (Newman-Keuls ANOVA), the difference between mature males and females was significant for DDE and PCB ($p < 0.01$), and t-nonachlor ($p < 0.05$); other statistical comparisons are shown in Table 10. The highest concentration of residues was in a mature male with 1.3% lipid in blubber; PCB was 6800 ppm, DDE 2000 ppm, and t-nonachlor 400 ppm. There was a significant correlation (linear regression $p < 0.001$) of PCB with DDE, PCB with t-nonachlor, and DDE with t-nonachlor in blubber of all animals. The blubber of captive dolphins had PCB levels comparable to those of the immature animals; DDE and t-nonachlor levels were comparable to those in mature males. The blubber lipid of the other cetacean species had significantly lower PCB than the stranded dolphins; there was no consistent pattern for the other contaminants.

The concentrations of contaminants in liver lipids of T. truncatus (Table 8) had a pattern similar to that in blubber. Levels of DDE were lower in mature females than in immature ($p < 0.05$) and mature males ($p < 0.01$). The captive dolphins had higher DDE levels than the average for the stranded group as a whole. Mature females also had lower values for t-nonachlor than immature males ($p < 0.05$). The male with the highest levels of organochlorines in blubber also had the highest concentrations in liver - 5200, 1300, and 200 ppm for PCB, DDE, and t-nonachlor, respectively. These data were omitted from statistical computations so as not to skew the population mean. In the stranded pilot whales, PCB concentrations were below detectable limits, and DDE and t-nonachlor were significantly lower than in all but the mature female dolphins. As in blubber there were significant correlations ($p < 0.001$) among all three classes of compounds in all groups of animals.

In the stranded dolphins, concentration of residues in liver lipid did not correlate with the amount of extractable lipid from that organ (Fig. 2). However, none of the dolphins with liver lipid concentrations greater than 15% had PCB concentrations above 200 ppm, whereas those with less than 15% liver lipid had up to 750 ppm.

Liver and blubber residues in individuals were compared to assess the capacity of the liver to process the compounds. Three patterns were evident. 1) For PCBs, a number of dolphins had higher concentrations in liver than in blubber, indicating that liver was not eliminating compounds at the same rate at which they were being delivered from the blubber (Fig. 3). 2) DDE residues in some animals were higher in liver than in blubber, perhaps for the same reason, but also because liver metabolizes DDTs to DDE, and therefore contributes to the DDE load at that site (Fig. 3). 3) Only two individuals had higher t-nonachlor in liver than in blubber, suggesting that liver can process it as it is delivered. In fact, the compound was undetected in many liver samples, perhaps indicating its rapid metabolism or excretion.

Liver and blubber from 11 dolphins were analysed for individual PCB congeners. The representative distribution of each congener was similar in both tissues, and consistent with findings from other studies (Muir et al. 1988). Generally, congeners 138, 153 and 201 were the most highly concentrated. Excluding from the sample one dolphin with the lowest extractable blubber lipid, liver concentrations were 1.6 to 38 ppm, 2.9 to 43 ppm, and 0.3 to 27 ppm for congeners 138, 153, and 201, respectively; those in blubber were 2 to 75 ppm, 2.7 to 100 ppm, and 1.3 to 18 ppm, respectively. Within individual dolphins, the ratio of 138/153 was consistently and significantly ($p < 0.001$) higher in liver than in blubber. This pattern might be attributed either to more rapid mobilisation of 138 from the blubber, or reduced ability to clear it from the liver.

Brain samples from 18 stranded animals were analyzed for organochlorine residues using the described technique. Concentrations (wet weight) of PCB, DDE, and t-nonachlor were 0-4 ppm, 0-0.4 ppm, and 0-0.3 ppm, respectively, and did not correlate with levels in other tissues. Values were consistent with or lower than reported for other marine mammals (O'Shea et al. 1980).

Toxicology - Heavy Metals

Liver concentrations of heavy metals are presented on Table 11. No significant differences were noted in comparisons among immature and mature, and male and female dolphins. As in other species, mercury and selenium levels were highly correlated (Muir et al. 1988); all values for heavy metals were comparable to those reported for other odontocetes (Honda et al. 1983, Muir et al. 1988).

DISCUSSION

This has been the most extraordinary saga of cetacean disease on record. Between the time the first dolphin stranded in New Jersey in June 1987, and the last on Florida's east coast eleven months later, over 740 animals died. The exact toll is not known, since almost certainly some animals were not recovered. However, Scott et al. (1988) estimated that 50% or more of the coastal migratory stock between Florida and New Jersey died during this period. Without a guiding precedent to help uncover the cause, it was necessary for the investigation to sweep a broad range of disciplines before settling on the eventual path to the probable solution. The two most likely potential causes for an outbreak of this kind were considered to be infectious disease and poisoning. After weighing evidence from 18 months of field and laboratory analyses, we have concluded that brevetoxin, the neurotoxin produced by the dinoflagellate Ptychodiscus brevis, probably was the proximate cause of this devastating event.

Early findings led the investigators away from microbial agents as the principal cause of death. There was no single pattern of illness that could be associated with a known pathogen, though it was clear that infectious agents contributed to and sometimes dominated the clinical picture. The first animals to come ashore on Virginia Beach in late summer clearly had been ill for some time, with a condition that ultimately affected skin, liver, and lung, and led to the accumulation of fluid in the abdominal and thoracic cavities. Meanwhile, in New Jersey, Drs. W. Medway (University of Pennsylvania) and D. Roscoe (New Jersey Division of Fish, Game and Wildlife) indicated in personal communication that carcasses there were in better condition and less affected with secondary bacterial infection. It appeared these differences were regional; dolphins coming ashore on Virginia Beach died in warmer waters heavily contaminated with opportunistic bacteria. Over 50% of the 21 species of potentially pathogenic bacteria isolated from 48 dolphins were of the genus Vibrio. These seemed to have been associated with some of the problems in skin and blood vessels that ultimately killed many of the animals but were not the primary cause of disease. The overwhelming nature of some of the infections, which probably arose in the lung, may have been related to immunoincompetence, the cause of which cannot be established. The depletion of lymphoid follicles in spleen, lymph nodes, and the intestine supports this suggestion.

Some dolphins also had viral infections. Eight had a skin condition characteristic of dolphin pox (Geraci et al. 1979), complete with suspicious inclusion bodies but in which no virus particles could be detected. In view of public sentiment expressed during the outbreak, it was comforting but not surprising to learn that none of the dolphins examined showed evidence of retroviruses, the group of viruses which is associated with Acquired Immune Deficiency Syndrome (AIDS) and whose counterparts in animals could have been a cause of reduced ability to fight normally harmless diseases. In any event, such viruses have a long latent period, and would not likely culminate in a single outbreak of disease. Dr. K. Somers is continuing to characterize the reovirus-like particles isolated from an ulcer on the palate of a dolphin. It is premature to comment on the serological titers to canine distemper virus, a morbillivirus, in six of 13 blood samples. Kennedy et al. (1988) have diagnosed morbillivirus infection and found distemper-like lesions in harbor porpoises (Phocoena phocoena) from the Irish Sea. We found no evidence of such infection nor was a morbillivirus detected using techniques suitable for its propagation. It is possible that the dolphins had been previously infected with a virus that escaped detection, or was no longer present at the time of the outbreak. A study must be undertaken to determine whether the virus or other antigen responsible for the serological reaction is widespread in dolphins and whether it is a pathogen. This calls for an examination of blood samples from a broad range of cetaceans, and an investigation into the nature of the antigen.

Geographic and temporal patterns of mortality also lacked the hallmark of infectious disease. During August 1987, at least 125 dolphins stranded dead along the Virginia coastline; nearly 50 came ashore in each of the months before and after. Others, according to fish-spotter pilot Mr. D. Thompson, were reported dead in small clusters at sea 18 miles from Cape May, NJ (August 21, 1987). To create such an overall pattern, an infectious agent would have had to be highly virulent -- causing acute disease across all ages and both sexes, spreading rapidly over a broad geographic range, and killing groups of animals without pause. Viruses and some bacteria introduced either by airborne transmission or through direct contact are capable of producing such havoc. Seals exposed on crowded rookeries have fallen victim to epizootics of influenza (Geraci et al. 1982), morbillivirus (Mahy et al. 1988, Osterhaus and Vedder 1988) and leptospirosis (Vedros et al. 1971). Yet, there is little to suggest that these or other contagious organisms could spread as explosively among cetaceans. Dolphins are more dispersed in an environment which, unlike air, solid substrate or even a closed body of water, would not readily support the transmission of such agents.

The accumulating evidence led us to consider a point source contaminant as the cause of mortality. This was also a subject of public concern, as reflected by a train of media reports that sewage and toxic wastes were being discharged in the New York Bight and Delaware Bay areas. We approached the Environmental Protection Agency to obtain information on permitted and illegal dumping of municipal and industrial wastes off the mid-Atlantic states, and submitted tissues for heavy metal and organochlorine contaminant analysis.

Levels of contaminants in the dolphins' blubber were found to be among the highest recorded for a cetacean (Gaskin et al. 1971, 1983, Aguilar 1983, Tanabe et al. 1984, Martineau et al. 1987, Muir et al. 1988). Unfortunately, it is not possible to compare the levels with those in other *T. truncatus* as the only study on this species employed a different technique (King 1987). To ensure that the high values were not an artefact of our methodology, we analyzed blubber and liver samples from pilot and humpback whales, and harbor porpoises, for which published data exist. Results of PCB, DDE, and t-nonachlor analyses on the pilot whales agree closely with the recent findings of Muir (1988) for the same species. Residues in the blubber of humpback whales (DDE and PCB) are comparable to those reported by Taruski et al. (1975). Our DDE and PCB values in the harbor porpoise are similar to or lower than Gaskin's et al. (1971, 1983). The values in *Tursiops* stand unreservedly among the highest in cetaceans - a commentary on the state of eastern coastal waters.

High organochlorine levels in *T. truncatus* were not restricted to the stranded group; the captives had concentrations similar to those in all but the stranded mature males. The results from the beach-cast specimens obviously reflect the levels of contaminants in the nearshore environment, where the dolphins accumulate these substances. The residues occur in the blubber of captives perhaps because they are given contaminated food, or more likely because with a steady diet, they have no need to mobilize blubber fat which would deliver the compounds to liver for excretion. Under these stable conditions, the presence of organochlorines in blubber may not pose a risk. Free-ranging animals facing intermittent food supply, or mobilizing fat during lactation, migration or times of illness, release compounds from this depot into vital, perhaps more critical organs such as liver.

Considering the evidence that at least some of the dolphins were mobilizing PCBs from blubber to liver, it is conceivable that blood levels rose and were sustained long enough to exert an effect. One class of organochlorines, the polychlorinated biphenyls (PCBs), can be harmful following both acute and chronic exposure (Safe 1985). Typically affected are liver and skin, and nervous, reproductive, and immune systems (Safe 1985). Yet we cannot categorically relate any of the conditions observed in the dolphins to the known effects of these compounds because of vast differences in response within and between species. Furthermore, it is unlikely that contaminants were the key to the event. The timing of the outbreak would have required that these compounds be mobilized to functionally toxic levels within a synchronized time-pulse. This is an unlikely scenario for substances which for decades have been a constant ingredient in their environment and body tissues, unless something else triggered their release by first debilitating the dolphins.

Biotoxins were considered to have this capability. The possibility was strengthened when saxitoxin, a neurotoxin produced by marine dinoflagellates, was found to be responsible for the deaths of 14 humpback whales, *Megaptera novaeangliae*, in early December 1987 and January 1988, in Cape Cod Bay (Geraci *et al.* submitted for publication). On the heels of that study, we analyzed liver samples from 17 dolphins that had died during the early, middle and late phases of the outbreak. There was no evidence of saxitoxin in these tissues.

By late summer 1988, some of the dolphin liver samples were reported to contain brevetoxin (PbTx), a lipid-soluble polyether toxin produced by the unarmored marine dinoflagellate *Ptychodiscus brevis*, Florida's red tide organism. The neurotoxin is extraordinarily potent, capable of generating effects in the nanomolar to picomolar concentration range *in vivo* (Baden, in press). When the analyses were completed in January, 1989, PbTx was found to be in the livers of eight of the 17 beached dolphins collected during the outbreak. No toxin was detected in any of the 17 controls, selected from dolphins that died in captivity, others in regions or at a time not related to the fatalities under investigation, and three that died during capture in October, 1987 (Table 6). A greater number of analyses would have added statistical weight to these findings. Yet the tests are time-consuming, and by this writing, 34 dolphin samples in addition to the fish specimens were all that could be processed. The pattern is nevertheless clear: 47% of the 17 diseased animals contained the toxin; all the rest did not.

Levels in dolphin liver ranged between 80-16,000 ng/g, and the calculated total amount in that organ was 0.08-14.7 mg. Assuming all the toxin was confined to liver, the total body burden would have been 2-290 µg/kg, comparable to or orders of magnitude higher than the 2.85 µg/kg level known to cause illness in man (McFarren *et al.* 1965). These values are conservative. Standard extraction procedures are only quantitative for one unaltered form of PbTx. Other forms that are covalently bound or otherwise modified were not considered. Nor is it reasonable to assume that all the toxin was in liver.

Signs of PbTx poisoning in fish and mammals are related to its action on the nervous system. Mice lose motor control, become paralysed and die of respiratory arrest (Baden and Mende 1982). The site of action is the voltage-sensitive sodium channel in excitable membranes, where the toxin causes increased sodium flux with subsequent depolarization and persistent activation of excitable cells (Poli *et al.*, in press). Death is rapid, and there are no reports of discernable histopathologic changes in acutely poisoned animals. Might this account for the presence of PbTx in a menhaden recently consumed by dolphin KDL-644 that showed no evidence of toxin in its liver?

Most of the dolphins did not die this way. They manifested an array of chronic disorders including fibrosis of liver and lung, adhesions of abdominal and thoracic viscera, and secondary microbial infections associated with immune suppression, as evidenced by histological changes in lymph nodes. We suggest that sublethal exposure to PbTx precipitated the train of events leading to some or all of these chronic changes. PbTx promotes peripheral vasodilation (Poli et al., in press) and is cardiotoxic (Rodgers et al. 1984). As a toxic aerosol, or once absorbed, it disrupts neural control of respiration (Borison et al. 1980) and induces bronchoconstriction (Baden et al. 1982). Symptoms of poisoning in humans reflect the gastrointestinal and neurologic action of the toxin. They include nausea, vomiting, diarrhea, reversal of temperature sensation, ataxia, and numbness and tingling of extremities (Baden 1983). A dolphin so affected would likely stop eating, eventually exhaust its blubber reserve, and thereby lose its passive buoyancy and thermal shield. The stress associated with these changes alone could set the stage for infection by the ubiquitous opportunistic organisms that were isolated from the affected dolphins. Superimposed on this, any direct neurotoxic effect of PbTx would be particularly threatening to a diving mammal.

How were dolphins exposed to the toxin? Red tides in southeastern U.S. waters normally originate 20-75 km west of the central Florida coast in the Gulf of Mexico (Steidinger and Haddad 1981), and generally remain within the Gulf where they eventually dissipate. Occasionally, as in 1972, 1977, and 1980 (Roberts 1979, Steidinger and Baden 1984), they can be entrained and transported to the east coast of Florida by the Gulf Loop Current-Florida Current-Gulf Stream system. This happened in the fall of 1987, and resulted in the eventual closure of shellfish beds along the North Carolina coast; there also were reports of respiratory and eye irritation in fishermen and residents (Tester et al. in press). Yet the toxin was found in the livers of dolphins that beached in Virginia three months before that time. They must have encountered the organisms sometime and somewhere along their northerly migration route.

In February, 1987, a P. brevis bloom was 25 km from a point where Gulf waters are transported to the east coast. Drift bottle data (Williams et al. 1977) suggest that a fragment could have reached the east coast by spring of that year. The possibility exists that blooms had been occurring all summer in and adjacent to the Gulf Stream, and went undetected until a filament reached the North Carolina coast in October, 1987. Such blooms would have been difficult to detect at sea, as they are not easily seen from vessels and there would have been little in the way of toxic aerosols, which are generally produced by waves and surf action in shallow waters. Planktivorous fish might have consumed the cells offshore during their migration northward. And dolphins could have obtained the toxin by eating these fish or their predators. These conditions would have exposed dolphins both directly in water, and indirectly in food, to PbTx for an extended period, with effects manifested a short time later as they reached the mid-Atlantic coast.

Brevetoxin was recovered from three yellowfin menhaden, B. smithii, caught off Vero Beach, FL, in late February 1988, and one unidentified menhaden taken from the stomach of a dolphin that stranded near Cape Canaveral on January 12, 1988. The finding of brevetoxin in fish at that time and place suggests that there was a persistent, undetected bloom that kept the food-web contaminated through the winter. Alternatively, the bloom that had delivered the filament to North Carolina in October 1987, had dissipated and left fish contaminated for at least three months. The first scenario challenges our understanding of the process of P. brevis blooms, the second of the dynamics of brevetoxin transfer in marine organisms.

In the fall of 1987, on their southerly migration, dolphins encountered the bloom off North Carolina. P. Tester (NOAA-NMFS Beaufort Laboratory, pers. comm.) observed dolphins surfacing in the blooms at that time. Three months later, and perhaps all along, they were feeding on contaminated fish. We believe that this second encounter with the toxin was responsible for the wave of stranded animals recovered along the Florida coast in the winter of 1987-1988; three of six dolphins examined had PbTx-2 in liver.

Levels of PbTx in the viscera of the live-caught menhaden translate to 200 μg of toxin per 500 g fish. Using this value, a dolphin feeding on menhaden at a rate of 10 kg each day, would consume 4 mg of PbTx. That is below the 6 mg/kg LD50 for mice, but if general toxicological dogma is applied, much lower doses would be required to incapacitate an animal as large as a dolphin. In fact, only 0.2 mg can cause illness in people.

Not all the dolphins were poisoned by eating fish. PbTx was found in the livers of three nursing calves. Dolphin WAM-295, with the highest concentration of PbTx in liver, was estimated to be less than 3 months of age. The toxin had to have been delivered in the milk, suggesting that like other lipid soluble residues, PbTx may be stored in fatty depots and mobilized along with fats as the animal draws on these reserves. There is no precedent for the finding of PbTx in milk, nor has this route of PbTx elimination been considered.

The circumstantial evidence suggests that PbTx is the most probable cause of the mortality. Contributing to the ultimate demise of the animals was a host of microbial and environmental factors. This is unlikely to have been the first time that dolphins have been exposed to the toxin. P. brevis blooms regularly occur on the Gulf coast of Florida. There they are restricted geographically in contrast to dolphins which move about freely. The chance of encounters is therefore reduced. They do occur, and at least one other associated mortality of dolphins has been reported (Gunter et al. 1948). Because there has been no search for biotoxins in stranded animals, other poisonings would have gone undiagnosed. One might also speculate that dolphins in the Gulf of Mexico have encountered blooms often enough to associate malaise with the ingestion of toxin-containing organisms or the aerosol, and thereafter avoid contact.

The episode along the east coast obviously required that the circumstances that delivered the organisms there be coupled with the presence of carrier-fishes situated in the path of migrating dolphins. The unparalleled scope of this event would suggest that all of these conditions have been met rarely, if at all, in the past. The summer of 1987 was unusual by any measure. In North Carolina, human poisoning from consumption of fish (Bonaventura and Bonaventura 1987) and shellfish (Tester et al. 1989) further attest to the unusual conditions that year.

The toxin in yellowfin menhaden has relevance to human health. Though not a fish that is commercially harvested, its southern range overlaps with related species of surface-feeding planktivores that are. In this case, the toxin was present in viscera and not the flesh, thus presenting no risk to humans consuming traditionally prepared fish, or the oils which are extracted under conditions that should destroy the toxin (Poli 1988). To establish whether a risk in fact exists, studies should be directed toward determining the uptake, distribution, persistence and transfer of PbTx in some representative commercially exploited species.

The discovery of PbTx in the dolphins and its previous circumstantial link to manatee deaths (O'Shea and Rathbun 1983) lead to a new generation of thought on factors contributing to natural mortality of marine mammals. Many questions will remain unanswered until directed studies are pursued. They must include: judicious examination of a representative sample of stranded marine mammals for biological toxins; studies on effects of chronic, sublethal exposure to PbTx; retrospective correlations between blooms and peak episodes of mortality; and determination of the environmental conditions that lead to the unusual event of 1987. Equally important is the need to resolve the growing question of whether contaminants at levels found in the dolphins might have affected their resilience and rendered them more susceptible either to the toxin or to the microorganisms that eventually brought them to their demise.

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Figure 1. Frequency distribution of the concentration of lipid extracted from blubber and liver of stranded bottlenose dolphins.

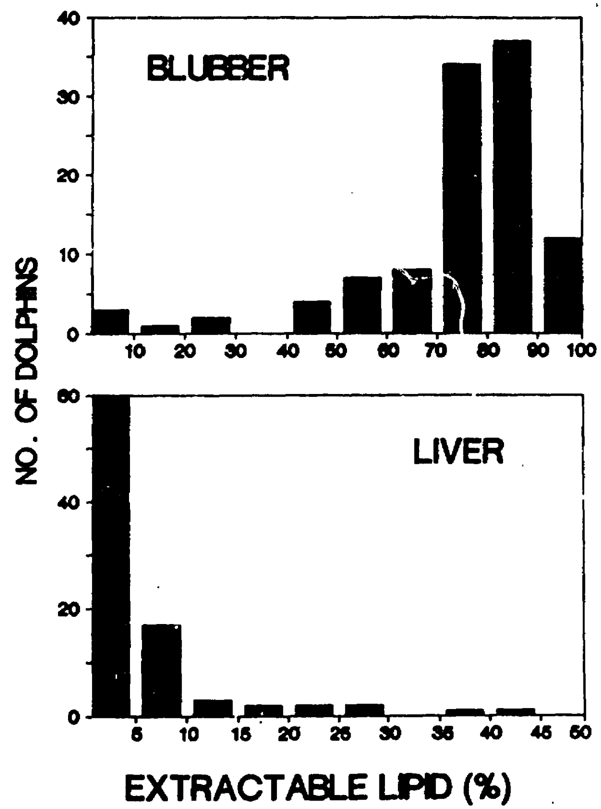

Figure 1

Figure 2. PCB concentrations in liver of stranded bottlenose dolphins, compared on the basis of lipid weight and wet weight as a function of lipid content in liver. None of the dolphins with liver lipid concentrations greater than 15% had PCB concentrations above 200 ppm.

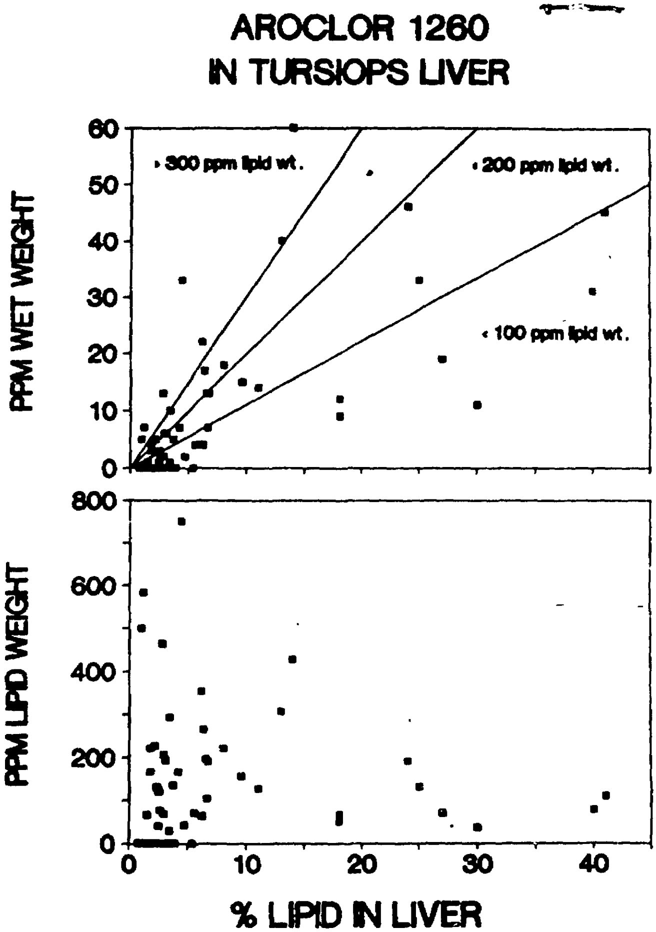
AROCLOR 1260
IN TURSIOPS LIVER
300 ppm lipid wt.
200 ppm lipid wt.
100 ppm lipid wt.
PPM WET WEIGHT
PPM LIPID WEIGHT
% LIPID IN LIVER
0
10
20
30
40
50
60
200
400
600
800

Figure 2

Figure 3. Comparison of the concentrations of three organochlorines in the liver and blubber of stranded bottlenose dolphins. Points lying above the line represent animals having greater concentrations in liver than in blubber, suggesting inability of the liver to clear the compounds.

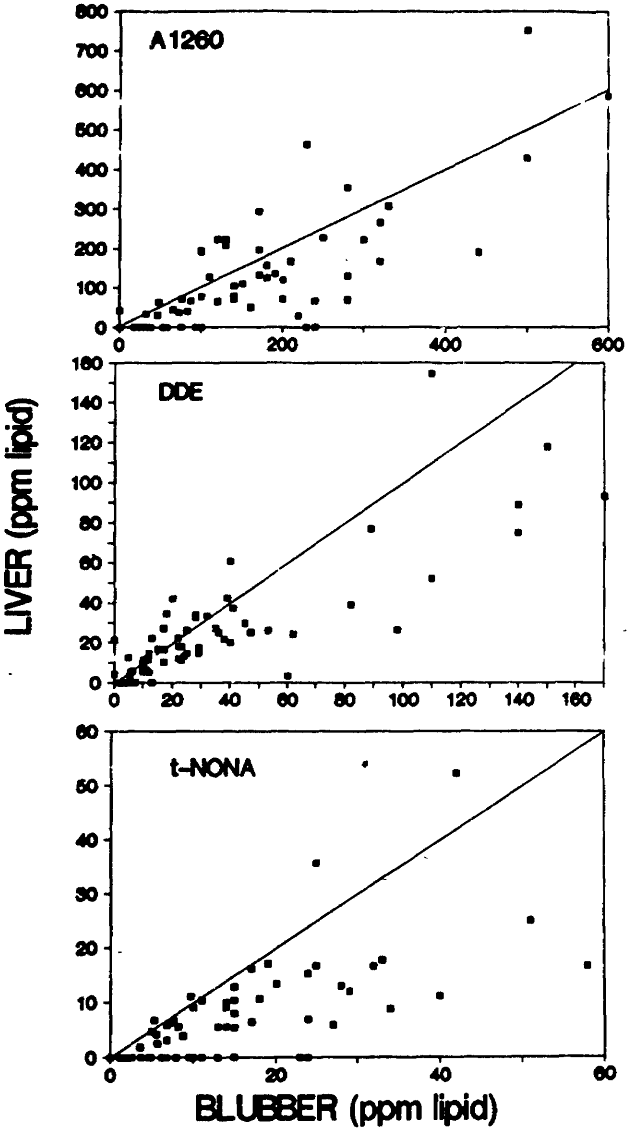
A1260
DDE
t-NONA
LIVER (ppm lipid)
BLUBBER (ppm lipid)
0
100
200
300
400
500
600
700
800
0
200
400
600
0
20
40
60
80
100
120
140
160
0
20
40
60
80
100
120
140
160
0
10
20
30
40
50
60
0
20
40
60

Figure 3

Table 1. Analytical disposition of 347 specimens collected during the 1987-88 mass mortality of bottlenose dolphins.

Analysis	No. of Dolphins	No. of Analyses
Partial necropsy	240	
Morphometric exam	61	
Complete necropsy	46	
Histopathology	95	2,660
Bacteriology	48	117
- *Chlamydia*	42	116
Virology	63	721
Toxicology		
- Organochlorine	75	1,456
- Heavy metals	68	1,079
Biotoxins		
- Water soluble	13	13
- Lipid soluble	34	34
Clinical pathology	26	1,106

Table 2. Tissues examined for evidence of viral infection.

Virus	Spleen	Liver	Kidney	Lung	Heart	Brain	Blood	Lymph node
Influenza A	7	9	7	9	4	3	1	1
Influenza B	7	9	7	9	4	3	1	12
Parainfluenza 1	7	9	7	9	4	3	1	12
Parainfluenza 3	7	9	7	9	4	3	1	12
Herpes 1	7	9	7	9	4	3	1	12
Herpes 2	7	9	7	9	4	3	1	12
Varicella- Zoster	7	9	7	9	4	3	1	12
Adenovirus	7	9	7	9	4	3	1	12
Papovirus	7	9	7	9	4	3	1	1
Bovine Leucosis	1				4		11	4
Bluetongue	1			4			11	4
Equine Infectious Anemia		1			4		9	4
Equine Rhinopneumonitis	1			3			9	4
Equine Herpes 2							13	
Coital Exanthema							15	
Ovine Progressive Pneumonia	1			4			9	4
Vesicular Stomatitis	1			4			9	4
Parvovirus							1	
Enterovirus							1	
AIDS virus							17	
Retrovirus				4			11	
Contagious Ecthema				3			11	

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Table 3: Gross necropsy findings in *Tursiops truncatus*. Observations reported in relation to the number of animals in which each organ system was examined.

OBSERVATION	OCCURRENCE OVERALL	MALE	FEMALE	MATURE	IMMATURE
lymph nodes					
necrosis	5/51	3/27	2/24	1/14	4/37
fibrosis	11/51	6/27	5/24	6/14[a]	5/37[a]
lymphadenitis	35/51	16/27	19/24	10/14	25/37
integument					
tattoo (pox)	6/119*	2/52	4/56	1/36	5/83
pock (craters, fissures)	6/119	3/52	3/56	5/36[b]	1/83[b]
ulcers	27/119	12/52	14/56	6/36	21/83
blisters (vesicles)	4/119	3/52	1/56	0/36	4/83
vascular lesions	6/119	1/52	5/56	2/36	4/83
necrosis	90/119	50/52	39/56	21/36	69/83
abdominal cavity					
adhesions	21/68	12/37	8/31	7/12	14/56
fluid - clear	8/68	6/37	2/31	3/12	5/56
fluid - sero-sanguineous	30/68	16/37	14/31	5/12	25/56
serosal fibrosis	7/68	3/37	4/31	3/12	4/56
thoracic cavity					
adhesions	14/51	9/30	5/20	5/12	9/39
fluid - clear	25/51	10/30	14/20	5/12	20/39
fluid - sero-sanguineous	34/51	24/30	10/20	7/12	27/39
pleural fibrosis	12/51	5/30	7/20	2/12	10/39
liver					
fibrosis	29/63	13/31	16/32	15/19	14/44
fatty (pale, yellow)	21/63	10/31	11/32	4/19	17/44
congestion	17/63	9/31	8/32	4/19	13/44
capsular fibrosis	14/63	8/31	6/32	5/19	9/44
degeneration	9/63	5/31	4/32	1/19	8/44
lung, pleura					
necrosis	40/113	20/55	20/58	7/27	33/86
edema	38/113	21/55	17/58	10/27	28/86
congestion	37/113	18/55	19/58	10/27	27/86
hemorrhage	11/113	7/55	4/58	2/27	9/86
fibrosis	18/113	7/55	11/58	5/27	13/86
parasitic pneumonia	22/113	12/55	10/58	3/27	19/86
interstitial pneumonia	40/113	16/55	24/58	11/27	29/86
bronchitis	6/113	1/55	5/58	1/27	5/86
pleura	26/113	13/55	13/58	8/27	18/86

[a] significantly different $p < 0.05$
[b] significantly different $p < 0.01$
* totals include 1 animal not sexed

Table 4: Histopathologic findings in *Tursiops*

OBSERVATION	OVERALL	MALE	FEMALE	MATURE	IMMATURE
	OCCURRENCE				
lymph node					
lymphadenitis	18/62*	10/28	8/33	10/20[a]	8/42[a]
follicle depletion	38/62	20/28	18/33	11/20	27/42
liver					
capsular fibrosis	16/67*	7/30	9/35	10/24[a]	6/43[a]
biliary fibrosis	7/67	5/30	2/35	4/24	3/43
parenchymal fibrosis	25/67	10/30	15/35	14/24[a]	11/43[a]
hepatitis	8/67	3/30	5/35	3/24	5/43
integument					
parakeratosis	15/62*	4/24	11/36	6/24	9/38
inclusions	8/62	5/24	3/36	3/24	5/38
ulcers (with dermatitis)	28/62	11/24	16/36	17/24[a]	11/38[a]
ulcers (without dermatitis)	1/62	1/24	0/36	0/24	1/38
lung					
pleura					
inflammation	5/77*	2/34	3/34	2/27	3/50
fibrosis	40/77	20/34	20/43	20/27[a]	20/50[a]
parenchyma					
mycotic infection	16/77	6/34	10/43	4/27	12/50
bacterial infection	10/77	4/34	6/43	5/27	5/50
parasitic infection	15/77	7/34	8/43	2/27	13/50
fibrosis	30/77	15/34	15/43	13/27	17/50
edema/congestion	14/77	6/34	8/43	5/27	9/50
bronchi/bronchioles					
desquamation	56/77	24/34	32/43	19/27	37/50
necrosis	10/77	6/34	4/43	2/27	8/50
inflammation	14/77	2/34	12/43	4/27[a]	10/50[a]
exudate	35/77	16/34	19/43	13/27	22/50
aspiration	10/77	3/34	7/43	1/27	9/50
vessel inflammation	2/77	1/34	1/43	1/27	2/50
heart					
myocardial fibrosis	16/54	6/23	10/31	7/16	9/38

[a] significantly different $p < 0.05$
* totals include animals not sexed

Table 5. Bacteria isolated from the tissues of 48 bottlenose dolphins examined during the mass mortality.

	Liver	Spleen	Lung	Lymph Node	Blood	Urine	Blubber	Abdom. Fluid	Kidney	Brain
No. of specimens	19	15	27	15	15	5	2	5	11	3
Vibrio sp.	12	9	26	13	8	2	2	1	7	3
V. parahaemolyticus	6	6	4	1	4		1		4	
V. damsela	2	1	6	1	1				1	1
V. alginolyticus	2	1	11	9	2		1		2	1
V. harveyi	2		1	2	1				4	
V. vulnificus	1		1			1		1		
V. nereis						1				
Edwardsiella sp.	8	6	10	3	1	1	1	3	3	
E. tarda	8	5	10	3	1	1	1	2	3	
E. hoshinae								1		
Alteromonas sp.	1	1	7	1	1	1	2	1		
A. putrefaciens	1	1	6	1	2	1	2	1		
Pseudomonas putrefaciens	1	1	2		2		1		1	
Enterobacter cloacae	1	1		1				1		
Acinetobacter lwoffi	1	1				1	1			
Streptococcus sp.	6		1	1		1			3	1
S. equisimilus	2	2	1							2
Escherichia coli	2	1	1	1					1	
Staphylococcus sp.	1	1	1	1	3		1			
Proteus sp.	1	1				1				
Morganella morganii		1								

Table 6: Results of brevetoxin analysis in dolphin liver samples.

SAMPLE	BIOASSAYS 1st	2nd	3rd	HPLC	CONC ng/g
Stranded, Virginia, Aug. 1987					
WAM 239	+	+	+	+	93
WAM 231	+	+	+	+	83
WAM 226	+	+	+	*	
WAM 214	+	+	+	**	
WAM 219	+	+	+	**	
JGM 448	+	-			
Stranded, Virginia, Sept.-Oct. 1987					
WAM 295	+	+	+	+	15820
WAM 280	+	+	+	+	14530
WAM 296	+	+	+	+	1851
WAM 282	+	+	+	**	
CWP 273	+	-			
Stranded, Florida, Jan.-Feb. 1988					
S-88-TT-51	+	+	+	+	14700
S-88-TT-57	+	+	+	+	310
S-88-TT-01	+	+	+	+	155
S-88-TT-11	+	+	+	*	
K 644	+	+	+	*	
SS-88-TT-04	+	+	+	**	
Died during capture, VA Beach, Oct. 1987					
VB-87-004	+	+	+	*	
VB-87-014	+	+	-		
VB-87-009	+	+	-		
Stranded - Texas, 1987-1988					
C 552	+	+	+	*	
C 391	+	-			
C 575	+	-			
Stranded mid-Atlantic Coast, Aug.-Nov. 1988					
WAM 331	+	+	-		
WAM 336	+	+	-		
WAM 340	+	+	-		
WAM 332	+	+	-		
WAM 335	+	+	-		
WAM 339	+	-			
Captive Tursiops					
MH82222 L21	+	+	-		
MH7408 L22	+	-			
MH7516	-				
MH79179	-				
Stranded - Cape Cod 1983					
MH83216	-				

* peak present but did not comigrate with standard
** no peak suggestive of PbTx

Table 7. Chlorinated hydrocarbon residues (as ppm lipid weight) in blubber from bottlenose dolphins recovered during the mass mortality. Samples from captive dolphins, pilot whales and harbour porpoise were analyzed concurrently for comparison.

SPECIMEN	N	Aroclor 1260 x ± SD	Aroclor 1260 RANGE	DDE x ± SD	DDE RANGE	Trans-Nonachlor x ± SD	Trans-Nonachlor RANGE	% Lipid x ± SD	% Lipid RANGE
Tursiops	56	181.6 ± 141.4	13-620	39.5 ± 44.7	3-200	14.6 ± 1[illegible]	1-58	78.3 ± 10.0	50-99
Imm. Female	18	145.1 ± 126.6	29-500	28.6 ± 37.1	6-170	15.3 ± 12.1	1-51	81.0 ± 7.7	63-96
Mat. Female	9	122.8 ± 96.5	18-280	14.2 ± 14.9	3-53	7.4 ± 8.4	1-28	73.3 ± 9.0	58-96
Imm. Male	22	202.3 ± 139.5	33-600	36.6 ± 31.6	8-140	16.8 ± 13.3	1-58	79.4 ± 9.9	54-92
Mat. Male	6	328.3 ± 140.9	100-620	114.5 ± 49.0	45-200	20.7 ± 5.3	13-28	69.1 ± 9.3[a]	50-79
Captive	3	177.7 ± 110.1	33-300	106.0 ± 55.3	28-150	18.4 ± 10.9	5-32	78.7 ± 4.5	75-85
Globicephala melaena	11	26 ± 20	10-69	22.1 ± 19.2	6-70	6.6 ± 3.8	4-18	82.5 ± 6.0	72-94
Phocoena phocoena	8	24 ± 6	15-33	8.2 ± 2.9	5-14	7.8 ± 2.6	5-12	89.3 ± 5.4	82-95
Megaptera novaeangliae	8	13 ± 12	6-44	4.5 ± 4.9	1-17	1.5 ± 2.0	0.2-7	73.0 ± 9.8	50-89

Table 8. Chlorinated hydrocarbon residues (as ppm lipid weight) in liver from bottlenose dolphins recovered during the mass mortality. Samples from captive dolphins, pilot whales and harbour porpoise were analyzed concurrently for comparison.

SPECIMEN	N	Aroclor 1260		DDE		trans-Nonachlor		% Lipid	
		$\bar{x} \pm SD$	RANGE	$\bar{x} \pm SD$	RANGE	$\bar{x} \pm SD$	RANGE	$\bar{x} \pm SD$	RANGE
Tursiops	53	145.7 ± 161.6	0-750	24.5 ± 26.5	0-155	8.1 ± 9.5	0-52	7.4 ± 9.5	0.8-41
Imm. Female	21	115.1 ± 96.7	0-429	20.4 ± 19.4	3-93	7.6 ± 6.4	0-25	11.2 ± 11.7	0.9-41
Mat. Female	11	72.4 ± 101.6	0-294	8.2 ± 8.9	0-26	2.5 ± 4.4	0-13	2.2 ± 0.8	0.8-3
Imm. Male	17	205.2 ± 214.1	0-750	35.4 ± 34.5	7-155	12.4 ± 13.2	0-52	6.7 ± 8.3	0.9-30
Mat. Male	4	254.4 ± 165.6	75-500	44.0 ± 19.4	30-77	7.4 ± 5.6	0-15	5.2 ± 4.6	1.0-13
Captive	3	109.2 ± 81.4	34-222	30.2 ± 34.8	34-118	11.4 ± 4.1	7-17	3.5 ± 1.8	1.8-5.9
Globicephala melaena	11	not detected		5.3 ± 10.8	0-38	1.6 ± 4.4	0-15	2.5 ± 1.5	1.0-5.7
Phocoena phocoena	9	46.2 ± 38.2	0-111	5.1 ± 3.8	0-11	3.3 ± 3.2	0-8.7	3.6 ± 1.6	1.6-6.2

Table 9. Chlorinated hydrocarbon residues (as ppm wet weight) in liver from bottlenose dolphins recovered during the mass mortality. Samples from captive dolphins, pilot whales and harbour porpoise were analyzed concurrently for comparison.

SPECIMEN	N	Aroclor 1260 x ± SD	Aroclor 1260 RANGE	DDE x ± SD	DDE RANGE	Trans-Nonachlor x ± SD	Trans-Nonachlor RANGE	% Moisture x ± SD	% Moisture RANGE
Tursiops	53	10.3 ± 13.7	0-60	1.7 ± 2.6	0-13	0.8 ± 1.2	0-5.5	71.6 ± 7.7	43-81
Imm. Female	21	14.2 ± 17.4	0-60	2.4 ± 3.3	0.2-13	1.2 ± 1.7	0-5.5	67.9 ± 9.2	43-79
Mat. Female	11	2.0 ± 1.0	0-10	0.2 ± 0.2	0-0.6	0.1 ± 0.1	0-0.3	76.5 ± 2.2	72-81
Imm. Male	17	10.2 ± 9.1	0-33	1.7 ± 1.6	0.1-6.9	0.8 ± 0.8	0-2.7	72.5 ± 6.6	55-30
Mat. Male	4	13.0 ± 15.6	2-40	3.1 ± 4.0	0.3-10	0.6 ± 0.8	0-2.0	73.9 ± 2.2	70-76
Captive	3	2.7 ± 0.9	2-4	2.3 ± 0.7	1.6-3.3	0.3 ± 0.05	0-0.4	74.7 ± 1.2	73-76
Globicephala melaena	11	not detected		0.14 ± 0.23	0-0.3	0.03 ± 0.06	0-0.2	74.1 ± 4.5	63-78
Phocoena phocoena	9	1.8 ± 1.5	0-4	0.2 ± 0.16	0-0.5	0.14 ± 0.13	0-0.4	72.6 ± 2.3	71-78

Table 10. Statistical comparison [a] of organochlorine residues in blubber of stranded *Tursiops*.

	Arochlor 1260		
	Immature Males	Immature Females	Mature Females
Mature Males	*	**	**
Immature Males	-	n.s.	n.s.
Immature Females	-	-	n.s.

	DDE		
	Immature Males	Immature Females	Mature Females
Mature Males	**	**	**
Immature Males	-	n.s.	n.s.
Immature Females	-	-	n.s.

	trans-Nonachlor		
	Immature Males	Immature Females	Mature Females
Mature Males	n.s.	n.s.	*
Immature Males	-	n.s.	n.s.
Immature Females	-	-	n.s.

[a] comparisons made using Newman-Keuls ANOVA. n.s. - not significant; *, ** - $p < 0.05, 0.01$.

Table 11. Heavy metal analysis of liver [illegible]cted from bottlenosed dolphins sampled during the mass mortality. Specimens from captive dolphins, pilot whales, and harbor porpoises were analyzed concurrently for comparison. Data expressed as ppm wet weight.

Specimen	N	Copper x ± SD	Copper RANGE	Zinc x ± SD	Zinc RANGE	Lead x ± SD	Lead RANGE
Tursiops	59	8.3 ± 5.7	0.08-2[illegible]	76 ± 43	16-210	0.23 ± 0.67	0-3.1
Imm.Female	22	8.9 ± 6.[illegible]	[illegible].4-2[illegible]	89 ± 33	25-170	0.14 ± 0.43	0-1.6
Mat.Female	14	8.5 ± 5.[illegible]	[illegible].6-2[illegible]	39 ± 1[illegible]	22-[illegible]	0.11 ± 0.41	0-1.6
Imm.Male	18	6.9 ± 4.3	[illegible].1-1[illegible]	92 ± 44	16-210	0.33 ± 0.77	0-2.6
Mat.Male	5	10 ± 5	3-1[illegible]	68 ± 53	25-170	0.62 ± 1.24	0-3.1
Captive	3	15.5 ± 8.6	6.4-2[illegible]	58 ± 12	42-[illegible]8	1.7 ± 1.55	0-4.0
Globicephala melaena	11	11.8 ± 5.6	4.5-2[illegible]	78 ± 54	42-[illegible]10	1.2 ± 1.5	0-4.1
Phocoena phocoena	9	13 ± 8.4	5.1-3[illegible]	64 ± 33	25-145	0.17 ± 0.47	0-1.5

Table 11, cont'd. Heavy metal analysis of liver collected from bottlenosed dolphins sampled during the mass mortality. Specimens from captive dolphins, pilot whales, and harbor porpoises were analyzed concurrently for comparison. Data expressed as ppm wet weight.

Specimen	N	Cadmium		Selenium		Mercury	
		x ± SD	RANGE	x ± SD	RANGE	x ± SD	RANGE
Tursiops	59	not detected		9 ± 12	0-51	22 ± 27	0-110
Imm.Female	21	not detected		2 ± 4	0-12	5 ± 7	0-25
Mat.Female	14	not detected		22 ± 14	5-51	55 ± 26	2[illegible]-10
Imm.Male	18	not detected		4 ± 7	0-24	12 ± 19	0-75
Mat.Male	5	not detected		15.9 ± 8	5.3-29	32 ± 16	1[illegible]-59
Captive	3	not detected		not detected		12 ± 11	[illegible]-28
Globicephala melaena	11	15 ± 11	0-35	12 ± 13	0-37	29 ± 36	0-10[illegible]
Phocoena phocoena	9	0.5 ± 1.3	0-4.2	not detected		1.6 ± 1.6	0-3.[illegible]

Appendix 1. Data on <u>Tursiops</u> <u>truncatus</u> examined during the clinical investigation.

Dolphin ID	Stranding Date	Location	Age[1]	Length (cm)	Sex	CHC[2]	Heavy Metals	Necropsy[3]	Histo-Pathol	Virol.	Micro-biol.
S-87-TT-02	01-07-87	Ormond Beach FL	-[4]	122	F	-	-	P	-	-	-
S-87-TT-04	01-10-87	Painter's Hill FL	-	201	F	-	-	P	-	-	-
S-87-SP-06	02-03-87	Pte Verda Beach FL	-	217	M	-	-	P	-	-	-
S-87-TT-07	03-11-87	Pte Verda Beach FL	-	122	M	-	-	P	-	-	-
S-87-TT-08	03-11-87	South Ponte Verda Beach FL	-	122	M	-	-	P	-	-	-
S-87-TT-09	04-23-87	South Ponte Verda FL	-	167	-	-	-	P	-	-	-
WAM-139	05-15-87	Norfolk VA	I	205	F	-	-	P	-	-	-
WAM-144	05-21-87	Virginia Beach VA	M	247	F	-	-	P	-	-	-
WAM-147	05-26-87	Ship Shoal Is. VS	I	115	F	-	-	P	-	-	-
WAM-148	05-26-87	Hog Island VA	-	112	M	-	-	P	-	-	-

[1]Dolphins are classified as mature (M) or immature (I) based on examination of reproductive organs and/or vertebral epiphyses. Age data expressed as year class, determined by tooth layer counts (S. Hersh, NMFS); p - perinate (<3 months).

[2]Chlorinated hydrocarbon analyses.

[3]C -complete necropsy; P - partial necropsy; ND - no data; LC - live capture.

4(+) = analysis performed; (-) = no analysis performed.

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
WAM-142	05-29-87	Little Creek VA	-	108	F	-	-	-	-	-	-
JGM-446	06-06-87	Seaford VA	-	252	M	-	-	P	-	-	-
S-87-TT-12	06-07-87	Staug Beach FL	-	202	F	-	-	P	-	-	-
WAM-151	06-08-87	Virginia Beach VA	-	102	F	-	-	P	-	-	-
WAM-152	06-08-87	Frisco NC	-	254	F	-	-	P	-	-	-
WAM-153	06-08-87	Assateague Island MD	-	214	M	-	-	P	-	-	-
WAM-155	06-17-87	Cape Charles VA	-	215	M	-	-	-	-	-	-
WAM-154	06-18-87	Cape Charles VA	-	205	M	-	-	-	-	-	-
WAM-161	06-25-87	Assateague Island MD	-	105	M	-	-	P	-	-	-
WAM-160	06-27-87	Assateague Island MD	-	228	M	-	-	P	-	-	-
WAM-158	06-28-87	Cape Charles VA	-	230	M	-	-	-	-	-	-
WAM-159	06-28-87	Cape Charles VA	-	238	M	-	-	P	-	-	-
WAM-156	06-29-87	Penney's Hill VA	-	246	-	-	-	P	-	-	-
WAM-157	06-29-87	False Cape VA	-	249	F	-	-	P	-	-	-
WAM-166	07-02-87	Bellehaven VA	-	167	F	-	-	-	-	-	-
WAM-163	07-06-87	Fort Story VA	-	210	F	-	-	-	-	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
WAM-165	07-06-87	Mathews County VA	23	281	M	-	-	P	-	-	-
WAM-162	07-07-87	Ocean City MD	1	250	F	-	-	P	-	-	-
WAM-164	07-08-87	Hampton VA	8	241	M	-	-	P	-	-	-
WAM-167	07-08-87	Assateague Island MD	-	221	F	-	-	P	-	-	-
WAM-168	07-11-87	Virginia Beach VA	-	202	M	-	-	P	-	-	-
WAM-169	07-11-87	Virginia Beach VA	-	199	F	-	-	P	-	-	-
WAM-170	07-11-87	Haven Beach VA	14	270	M	-	-	P	-	-	-
WAM-190	07-12-87	Assateague Island MD	1	175	F	-	-	-	-	-	-
WAM-171	07-13-87	Seaford VA	-	219	M	-	-	P	-	-	-
WAM-172	07-13-87	Virginia Beach VA	[illegible]	-	-	-	-	P	-	-	-
WAM-173	07-16-87	Sandbridge VA	1	183	F	-	-	-	-	-	-
WAM-174	07-16-87	Sandbridge VA	16	269	M	-	-	-	-	-	-
CWP-249	07-17-87	Worcester MD	7	258	M	-	-	-	-	-	-
WAM-175	07-17-87	Lynn Haven Inlet VA	9	240	F	-	-	P	-	-	-
WAM-179	07-17-87	False Cape VA	-	220	F	-	-	P	-	-	-
CWP-251	07-18-87	Milford Haven VA	3	222	M	-	-	P	-	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
WAM-188	07-18-87	Hampton VA	-	245	F	-	-	-	-	-	-
WAM-176	07-19-87	Virginia Beach VA	1'	241	F	-	-	P	-	-	-
WAM-178	07-21-87	Hampton VA	9	267	M	-	-	-	-	-	-
WAM-180	07-21-87	Virginia Beach VA	I	220	-	-	-	P	-	-	-
WAM-181	07-21-87	Norfolk VA	2'	274	M	-	-	-	-	-	-
WAM-182	07-21-87	Norfolk VA	1'	255	M	-	-	-	-	-	-
WAM-184	07-22-87	False Cape VA	-	-	-	-	-	P	-	-	-
JGM-448	07-23-87	Assateague Island MD	5	238	M	-	-	P	-	-	-
WAM-186	07-23-87	Hampton VA	I	211	M	-	-	P	-	-	-
WAM-187	07-24-87	Hampton VA	9	254	F	-	-	-	-	-	-
WAM-189	07-28-87	Virginia Beach VA	-	254	M	-	-	P	-	-	-
WAM-191	07-29-87	Little Creek VA	4	233	F	-	-	P	-	-	-
WAM-192	07-29-87	Virginia Beach VA	2	202	M	-	-	P	-	-	-
WAM-194	07-29-87	False Cape VA	-	-	-	-	-	-	-	-	-
WAM-195	07-29-87	Ocean City MD	-	183	-	-	-	P	-	-	-
WAM-193	07-30-87	False Cape VA	11	284	M	-	-	P	-	-	-
WAM-196	07-30-87	Assateague Island MD	I	216	-	-	-	P	-	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
WAM-197	07-31-87	Croaton Beach VA	[illegible]3	270	F	-	-	P	-	-	-
WAM-198	07-31-87	Deacroke Island	14	270	M	-	-	P	-	-	-
CWP-252	08-02-87	Worcester MD	17+	259	F	-	-	P	-	-	-
WAM-199	08-02-87	Norfolk VA	8	242	F	-	-	-	-	-	-
WAM-200	08-02-87	Virginia Beach VA	[illegible]	237	M	-	-	P	-	-	-
WAM-201	08-02-87	Virginia Beach VA	[illegible]	212	M	-	-	P	-	-	-
WAM-203	08-02-87	Gwynn Island VA	1	-	-	-	-	P	-	-	-
WAM-213	08-02-87	Little Creek VA	[illegible]	222	F	-	-	-	-	-	-
WAM-202	08-03-87	Virginia Beach VA	1	206	M	-	-	P	-	-	-
CWP-253	08-04-87	Hampton VA	[illegible]	233	F	-	-	P	-	-	-
CWP-254	08-04-87	Hampton VA	8	236	F	-	-	-	-	-	-
CWP-255	08-04-87	Virginia Beach VA	1	203	F	-	-	P	-	-	-
CWP-256	08-04-87	Virginia Beach VA	4	223	M	-	-	P	-	-	-
WAM-204	08-04-87	Hampton VA	-	140	M	-	-	P	-	-	-
S-87-TT-14	08-06-87	Mayport FL	-	272	M	-	-	P	-	-	-
WAM-205	08-06-87	Damneck VA	[illegible]	193	M	-	-	C	-	-	-
WAM-206	08-06-87	Virginia Beach VA	13	242	M	-	-	C	+	+	+
WAM-207	08-06-87	Ocean Park VA	[illegible]	214	M	-	-	C	+	+	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
WAM-233	08-06-87	Back Bay VA	M	260	-	-	-	P	-	-	-
WAM-133	08-07-87	Norfolk VA	1y	154	M	-	-	-	-	-	-
WAM-208	08-07-87	Damneck VA	20+	303	M	-	-	P	+	+	+
WAM-216	08-07-87	Croatan Beach VA	-	-	-	-	-	-	-	-	-
WAM-217	08-07-87	Virginia Beach VA	4	241	F	-	-	-	-	-	-
S-87-TT-15	08-08-87	Mayport FL	M	270	M	-	-	P	-	-	-
WAM-209	08-08-87	Virginia Beach VA	1	188	F	+	+	C	+	+	+
WAM-210	08-08-87	Sandbridge VA	1y	159	F	+	+	C	+	+	+
WAM-211	08-08-87	Little Creek VA	5	197	F	-	-	P	-	-	-
WAM-212	08-08-87	Little Creek VA	-	164	F	-	-	P	-	-	-
WAM-214	08-08-87	Virginia Beach VA	-	242	F	-	-	C	+	+	+
WAM-215	08-09-87	Virginia Beach VA	-	240	F	-	-	P	-	-	-
WAM-218	08-09-87	Cape Henry VA	1y	148	M	-	-	P	-	-	-
VA-1	08-10-87	Virginia	I	-	-	+	+	-	-	+	+
WAM-219	08-10-87	Virginia Beach VA	-	134	M	-	-	P	-	-	-
70-87	08-11-87	Avalon NJ	-	267	F	-	-	-	+	+	+
72-87	08-11-87	Island Beach NJ	-	274	M	-	-	-	+	+	+
74-87	08-11-87	Long Beach Isl NJ	-	290	M	-	-	-	+	+	+

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
76-87	08-11-87	Island Beach NJ	[illegible]	224	M	+	+	-	+	+	+
CWP-260	08-11-87	Virginia Beach VA	[illegible]	239	F	-	-	P	-	-	-
DAP-011	08-11-87	Seashore State Park VA	1p	155	M	-	-	P	-	-	-
DAP-013	08-11-87	Lynn Haven Inl VA	[illegible]	232	M	-	-	P	-	-	-
WAM-220	08-11-87	Virginia Beach VA	[illegible]	220	-	+	-	P	+	-	-
WAM-221	08-11-87	Virginia Beach VA	[illegible]	184	F	-	-	P	-	-	-
WAM-222	08-11-87	Virginia Beach VA		140	F	-	-	P	-	-	-
WAM-223	08-11-87	Seashore State Park VA	[illegible]	137	F	-	-	P	-	-	-
WAM-234	08-11-87	Back Bay VA		199	M	-	-	-	-	-	-
CWP-257	08-12-87	Virginia Beach VA	1p	143	F	-	-	P	-	-	-
CWP-258	08-12-87	Virginia Beach VA	1	160	-	-	-	P	-	-	-
CWP-259	08-12-87	Virginia Beach VA	[illegible]	229	F	-	-	-	-	-	-
WAM-224	08-12-87	Seashore State Park VA	[illegible]	177	F	-	+	P	-	-	-
WAM-225	08-12-87	Little Creek VA	[illegible]	250	M	+	-	C	-	-	-
WAM-226	08-13-87	Virginia Beach VA	[illegible]	243	F	+	+	C	+	+	+
WAM-227	08-13-87	Virginia Beach VA	[illegible]	212	M	+	-	C	+	+	+

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
WAM-228	08-13-87	Chesapeake Beach VA	13	252	F	-	-	P	-	-	-
WAM-229	08-13-87	Fort Story VA	-	181	F	-	-	P	-	-	-
CWP-261	08-14-87	Virginia Beach VA	1	194	M	-	-	C	-	-	-
CWP-262	08-14-87	Virginia Beach VA	21+	248	M	-	-	P	-	-	-
S-87-TT-16	08-14-87	St. Augustine Beach FL	-	188	-	-	-	P	-	-	-
WAM-230	08-14-87	Virginia Beach VA	1p	143	F	+	+	C	+	+	+
WAM-231	08-14-87	Chesapeake Beach VA	J	240	F	+	+	C	+	+	+
WAM-232	08-14-87	Chesapeake Beach VA	16+	260	F	+	+	C	+	-	+
WAM-237	08-14-87	Portsmouth VA	M	232	F	-	-	P	-	-	-
85-87	08-15-87	Brigantine NJ	-	-	-	-	-	-	-	+	+
CWP-263	08-15-87	Virginia Beach VA	4	251	M	-	-	C	+	+	+
CWP-264	08-15-87	Virginia Beach VA	2	205	F	+	+	C	+	+	+
CWP-265	08-15-87	Virginia Beach VA	1p	134	F	-	-	P	-	-	-
CWP-266	08-15-87	Norfolk VA	12	285	M	-	-	C	+	-	-
WAM-235	08-15-87	Hampton VA	10	257	M	-	-	C	+	-	-
87-01	08-16-87	Virginia Beach VA	-	-	-	-	-	LC	-	-	+

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
87-02	08-16-87	Virginia Beach VA	-	-	-	-	-	LC	-	-	+
87-03	08-16-87	Virginia Beach VA	-	-	-	-	-	LC	-	-	+
WAM-236	08-16-87	Virginia Beach VA	I	220	F	-	-	C	+	-	+
S-87-TT-17	08-16-87	Ponce Inlet FL	-	137	M	-	-	P	-	-	-
DAP-014	08-20-87	Virginia Beach VA	7	226	M	-	-	P	-	-	-
WAM-238	08-20-87	Little Creek VA	1p	144	F	-	-	P	-	-	-
WAM-239	08-20-87	Sandridge VA	I	143	F	+	+	C	+	+	+
WAM-243	08-20-87	Back Bay VA	-	-	-	-	-	-	-	-	-
CWP-267	08-21-87	Virginia Beach VA	J	201	M	-	-	C	+	-	-
CWP-269	08-21-87	Seashore State Park VA	1	212	M	-	-	P	-	-	-
WAM-240	08-21-87	Wash Woods VA	1	186	F	-	-	P	-	-	-
WAM-241	08-21-87	North Carolina	3	227	F	-	-	P	-	-	-
WAM-242	08-21-87	False Cape VA	5	255	-	-	-	P	-	-	-
S-87-SF-18	08-22-87	Ponte Vedra FL	-	231	M	-	-	P	-	-	-
104-87	08-23-87	Townsend Inlet NJ	-	253	M	-	-	-	-	+	+
WAM-244	08-23-87	Virginia Beach VA	-	-	-	-	-	-	-	-	-
WAM-245	08-23-87	Chesapeake Bay VA	I	197	F	-	-	C	-	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
WAM-246	08-23-87	Virginia Beach VA	1p	115	F	-	-	C	+	-	-
WAM-247	08-24-87	Sandbridge VA	1	167	M	+	+	C	+	+	+
WAM-249	08-24-87	False Cape VA	-	-	-	-	-	-	-	-	-
WAM-250	08-24-87	False Cape VA	2	207	F	-	-	P	-	-	-
S-87-TT-19	08-26-87	Flagler Beach FL	-	155	F	-	-	P	-	-	-
WAM-251	08-26-87	Sandbridge VA	J	232	M	+	+	C	+	+	+
WAM-252	08-29-87	Black Croatan Beach VA	1p	151	M	+	+	C	+	+	+
WAM-253	08-29-87	Fort Story VA	22	281	M	+	+	C	+	+	+
WAM-254	08-30-87	Damneck VA	2	194	M	+	+	C	+	-	-
WAM-255	08-30-87	Virginia Beach VA	I	190	F	+	+	C	+	+	+
JGM-450	08-31-87	Virginia Beach VA	20	260	F	-	-	C	+	+	+
WAM-257	09-01-87	Fort Story VA	1	159	F	-	-	P	-	-	-
WAM-258	09-01-87	Sandbridge VA	8	201	M	+	+	C	+	-	-
WAM-259	09-01-87	Virginia Beach VA	1	111	M	+	+	C	+	-	-
WAM-260	09-01-87	Virginia Beach VA	13	238	F	-	-	-	-	-	-
WAM-261	09-01-87	Croatan Beach VA	2	191	M	+	+	P	+	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
NVSL 87-44280	09-03-87	Brigantine NJ	-	-	M	-	-	-	+	+	-
WAM-262	09-03-87	Virginia Beach VA	1	145	M	-	-	P	-	-	-
WAM-263	09-04-87	Virginia Beach VA	1	190	F	-	+	C	+	+	+
WAM-264	09-04-87	Camp Pendelton VA	13	253	F	+	+	C	+	+	+
WAM-265	09-04-87	Lynhaven Inlet VA	8	220	M	-	-	C	+	-	-
WAM-266	09-05-87	Virginia Beach VA	5	220	M	-	-	P	-	-	-
WAM-267	09-05-87	Virginia Beach VA	1	172	F	-	-	P	-	-	-
WAM-268	09-05-87	Damneck VA	2	215	F	-	-	P	-	-	-
117-87	09-06-87	Avalon NJ	-	226	F	+	+	-	-	+	-
126-87	09-06-87	Ventnor NJ	I	175	M	+	+	-	-	+	-
WAM-269	09-06-87	Virginia Beach VA	2	203	F	+	+	P	+	-	-
WAM-270	09-07-87	Damneck VA	3	230	F	-	-	P	-	-	-
WAM-271	09-07-87	Virginia Beach VA	I	147	F	-	+	P	-	-	-
WAM-272	09-08-87	Croatan Beach VA	9	-	-	-	+	-	-	-	-
WAM-273	09-08-87	Fort Story VA	-	-	-	-	+	P	-	-	-
WAM-278	09-08-87	Curova Beach NC	I	201	M	-	-	P	-	-	-
WAM-274	09-09-87	Virginia Beach VA	I	198	M	-	-	P	-	-	-
WAM-275	09-09-87	Rescue VA	1p	153	F	-	-	P	-	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
WAM-276	09-10-87	Ragged Island VA	-	-	-	-	-	P	-	-	-
WAM-277	09-10-87	Back Bay VA	2	190	M	-	-	P	-	-	-
WAM-279	09-10-87	False Cape VA	-	-	-	-	-	-	-	-	-
DAP-012	09-12-87	Fort Story VA	4	222	F	-	-	P	-	-	-
CWP-270	09-14-87	Hampton VA	1p	131	M	-	-	P	-	-	-
JGM-451	09-14-87	Sandbridge VA	1p	156	F	-	-	P	+	-	-
WAM-280	09-18-87	Sandbridge VA	20+	258	M	-	-	P	+	-	-
WAM-281	09-18-87	Sandbridge VA	-	-	-	-	-	-	-	-	-
WAM-282	09-18-87	Virginia Beach VA	1p	157	M	-	-	P	-	-	-
JGM-452	09-19-87	Ocean City MD	7	253	M	-	-	P	-	-	-
WAM-287	09-19-87	Fort Story VA	1p	151	M	-	-	P	-	-	-
JGM-453	09-20-87	Ocean City MD	3	224	M	-	-	P	-	-	-
WAM-283	09-20-87	Norfolk VA	7	210	M	-	-	P	-	-	-
WAM-284	09-20-87	Back Bay VA	1p	144	M	-	-	P	-	-	-
WAM-285	09-21-87	Virginia Beach VA	4	240	M	-	-	P	-	-	-
WAM-286	09-21-87	Virginia Beach VA	8	249	-	-	-	P	-	-	-
WAM-288	09-22-87	Fort Story VA	4	212	M	-	-	P	-	-	-
WAM-289	09-23-87	Sandbridge VA	1	211	M	-	+	P	+	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
WAM-290	09-23-87	False Cape VA	14	-	-	-	-	-	-	-	-
WAM-291	09-23-87	Corova Beach NC	22	255	F	-	-	P	-	-	-
WAM-292	09-23-87	Virginia Beach VA	15	-	-	-	-	P	-	-	-
WAM-293	09-23-87	Virginia Beach VA	6	-	-	-	-	-	-	-	-
WAM-294	09-23-87	Corova Beach NC	I	147	M	-	-	-	-	-	-
CWP-271	09-25-87	Virginia Beach VA	I	212	F	+	+	P	+	-	-
CWP-272	09-25-87	Virginia Beach VA	10	148	F	+	+	P	+	-	-
CWP-273	09-27-87	Little Creek VA	15	244	F	+	+	P	+	-	-
CWP-268	09-29-87	Norfolk VA	-	-	-	-	-	-	-	-	-
CWP-274	09-29-87	Norfolk VA	12	277	M	-	-	P	+	-	-
WAM-295	09-29-87	Back Bay VA	10	142	F	-	-	P	+	-	-
WAM-296	09-29-87	Virginia Beach VA	-	138	F	-	-	P	+	-	-
WAM-297	09-29-87	Virginia Beach VA	22	271	M	-	-	P	-	-	-
WAM-298	10-01-87	Sandbridge VA	5	204	M	-	-	P	-	-	-
WAM-299	10-05-87	Virginia Beach VA	M	240	F	-	-	P	-	-	-
VB-87-001	10-05-87	Virginia Beach VA	-	259	F	-	-	LC	+	+	-
VB-87-002	10-05-87	Virginia Beach VA	-	233	M	-	+	LC	+	+	-
VB-87-003	10-05-87	Virginia Beach VA	-	289	M	-	-	LC	+	+	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
VB-87-004	10-05-87	Virginia Beach VA	M	249	F	+	+	C	+	+	-
VB-87-005	10-06-87	Virginia Beach VA	20+	-	F	+	+	C	+	+	-
VB-87-006	10-06-87	Virginia Beach VA	I	168	-	-	-	LC	-	+	-
VB-87-007	10-06-87	Virginia Beach VA	-	283	M	-	+	LC	-	+	-
VB-87-008	10-06-87	Virginia Beach VA	M	269	F	-	+	LC	+	+	-
VB-87-009	10-06-87	Virginia Beach VA	7	226	F	+	+	C	+	+	-
VB-87-010	10-07-87	Virginia Beach VA	-	236	F	-	+	LC	-	+	-
VB-87-011	10-07-87	Virginia Beach VA	-	250	F	-	+	LC	-	+	-
VB-87-012A	10-07-87	Virginia Beach VA	-	257	F	-	-	LC	-	+	-
VB-87-012B	10-07-87	Virginia Beach VA	15	259	M	+	+	C	+	+	-
VB-87-013	10-07-87	Virginia Beach VA	I	166	-	-	-	LC	-	+	-
VB-87-014	10-08-87	Virginia Beach VA	1	258	F	+	+	C	+	+	-
VB-87-015	10-08-87	Virginia Beach VA	M	239	F	-	+	LC	+	+	-
VB-87-016	10-08-87	Virginia Beach VA	M	243	F	-	+	LC	+	+	-
VB-87-017	10-08-87	Virginia Beach VA	M	255	M	-	+	LC	+	+	-
VB-87-018	10-08-87	Virginia Beach VA	M	239	M	-	+	LC	+	+	-
WAM-301	10-12-87	Virginia Beach VA	-	154	M	-	-	P	-	-	-
WAM-300	10-13-87	Sandbridge VA	1.5	135	F	-	-	P	-	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
WAM-302	10-17-87	False Cape VA	4	234	M	-	-	P	-	-	-
WAM-303	10-17-87	Seashore State Park VA	31+	244	F	-	-	P	+	+	-
WAM-305	10-17-87	Buxton NC	1[illegible]	162	F	-	-	P	-	-	-
CWP-274A	10-17-87	Eastville VA	2[illegible]	270	M	-	-	P	-	-	-
CWP-275	10-18-87	Hampton VA	9	236	M	-	-	P	-	-	-
WAM-304	10-18-87	Virginia Beach VA	-	188	M	-	-	P	-	-	-
WAM-309	10-19-87	Parrymore Island NC	2[illegible]	269	M	-	-	P	-	-	-
WAM-306	10-20-87	Pea Island NC	I	132	F	-	-	P	-	-	-
WAM-307	10-21-87	Corolla NC	8	246	-	-	-	P	-	-	-
WAM-308	10-21-87	Corolla NC	I	193	M	-	-	P	-	-	-
WAM-310	10-21-87	Assateague Island MD	2[illegible]	279	M	-	-	P	-	-	-
S-87-TT-22	11-26-87	American Beach FL	-	156	M	-	-	P	-	-	-
S-87-TT-23	11-30-87	Fernandina Beach FL	-	158	F	-	-	P	-	-	-
S-87-TT-24	11-30-87	Fernandina Beach FL	-	250	F	-	-	P	-	-	-
S-87-TT-25	12-07-87	Jacksonville Beach FL	-	245	M	-	-	P	-	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
S-87-TT-26	12-07-87	Fernandine Beach FL	-	237	F	-	-	P	-	-	-
S-87-TT-27	12-13-87	Ponte Verda Beach FL	-	243	M	+	+	P	-	-	-
S-87-TT-28	12-15-87	Mayport FL	-	180	F	+	+	P	-	-	-
S-87-TT-29	12-17-87	Mayport FL	-	172	F	+	+	P	-	-	-
S-87-TT-30	12-20-87	Crescent Beach FL	-	270	M	+	+	P	-	-	-
S-87-TT-31	12-21-87	American Beach FL	-	265	M	+	+	P	-	-	-
S-87-TT-32	12-21-87	American Beach FL	-	190	F	-	-	P	-	-	-
S-87-TT-33	12-21-87	Ponte Vedra FL	-	244	M	-	-	P	-	-	-
S-87-TT-34	12-21-87	Hannah Park FL	-	214	M	-	-	P	-	-	-
S-87-TT-35	12-21-87	Ponte Verda FL	-	240	M	-	-	P	-	-	-
S-87-TT-45	12-21-87	Johnson Beach FL	-	180	F	+	+	P	-	-	-
S-87-TT-46	12-21-87	Fernandine FL	-	180	F	-	-	P	+	-	-
S-87-TT-36	12-23-87	Mayport FL	-	103	M	-	-	P	-	-	-
S-87-TT-37	12-23-87	Guano State Park FL	-	252	M	-	-	P	-	-	-
S-87-TT-38	12-23-87	Flagler Beach FL	-	210	F	-	-	P	-	-	-
S-87-TT-39	12-24-87	Guano State Park FL	-	145	M	-	-	P	-	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
S-87-TT-40	12-24-87	Guano State Park FL	-	160	-	-	-	P	-	-	-
S-87-TT-43	12-24-87	Johnson Beach FL	-	173	F	-	-	P	-	-	-
S-87-TT-41	12-25-87	St. Augustine Beach FL	-	195	M	+	+	P	-	-	-
S-87-TT-42	12-25-87	Daytona Beach FL	-	248	M	+	+	P	-	-	-
S-87-TT-44	12-27-87	Johnson Beach FL	-	180	M	-	-	P	-	-	-
S-87-TT-47	12-28-87	Amelia Island FL	-	235	F	-	-	P	-	-	-
S-87-SP-48	12-30-87	Ormond FL	-	205	M	-	-	P	-	-	-
S-87-TT-50	12-30-87	New Smyrna FL	-	130	M	-	-	P	+	-	-
S-87-TT-49	12-31-87	Daytona Beach FL	-	210	F	-	-	P	+	-	-
S-87-TT-51	12-31-87	Beverly Beach FL	-	248	F	-	-	P	-	-	-
S-88-TT-01	01-01-88	Atlantic Beach FL	-	205	F	+	+	P	+	-	-
S-88-TT-02	01-01-88	Mayport Beach FL	-	215	M	-	-	P	-	-	-
S-88-TT-03	01-01-88	Johnson Beach FL	M	250	F	-	-	P	+	-	-
S-88-TT-04	01-02-88	Amelia Island FL	M	250	M	+	+	P	+	-	-
S-88-TT-05	01-03-88	Daytona Beach FL	-	-	M	+	-	P	-	-	-
S-88-TT-06	01-03-88	Atlantic Beach FL	-	136	M	+	-	P	-	-	-
S-88-TT-07	01-04-88	Daytona Beach FL	I	162	M	+	+	P	+	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
S-88-TT-14	01-04-87	St. John's Cty FL	-	272	M	-	-	P	-	-	-
S-88-TT-15	01-05-88	St. John's Cty FL	-	251	M	-	-	P	-	-	-
S-88-TT-09	01-07-88	Ormond FL	-	233	M	-	-	P	-	-	-
S-88-TT-10	01-08-88	St. John's Cty FL	-	194	F	+	+	P	+	-	-
SWF-TT-8802-B	01-08-88	Sebastian Inlet FL	-	175	M	+	+	P	-	+	+
SWF-TT-8803-B KDL:371	01-08-88	New Smyrna Ranger Station FL	-	183	F	+	+	P	+	+	+
S-88-TT-11	01-10-88	Volusia Cty FL	11	243	F	+	+	P	+	-	-
S-88-TT-12	01-10-88	Flagler Beach FL	1	206	M	+	+	C	+	-	-
S-88-TT-20	01-10-88	Hammock FL	-	130	F	+	-	-	-	-	-
S-88-TT-13	01-11-88	St. John's Cty FL	-	230	M	+	-	P	-	-	-
S-88-TT-16	01-11-88	Nassau FL	1p	139	M	+	-	-	-	-	-
S-88-TT-17	01-13-88	Ponte Vedra Beach FL	13	260	M	-	-	P	-	-	-
S-88-TT-18	01-13-88	St. John's Cty FL	1	131	F	+	-	-	-	-	-
S-88-TT-19	01-13-88	Fort Clinch State Park FL	19+	248	F	+	+	C	+	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
S-88-TT-21	01-13-88	Hammock FL	1p	-	-	-	-	-	-	-	-
SWF-TT-8805-B KDL:643	01-13-88	Brevard Cty FL	1	132	F	+	+	P	-	-	+
SWF-TT-8804-B KDL:644	01-13-88	Brevard Cty FL	-	188	F	+	+	C	+	+	-
S-88-TT-22	01-15-88	Volusia Cty FL	1	191	M	+	-	P	-	-	-
SWF-TT-8806-B KDL:893	01-15-88	Indian River Cty FL	-	181	F	+	+	P	+	+	+
S-88-TT-23	01-16-88	Daytona Beach FL	18	263	M	+	-	P	-	-	-
S-88-TT-24	01-16-88	Volusia Cty FL	3	207	-	-	-	P	-	-	-
S-88-TT-25	01-16-88	Ormond Beach Fl.	-	230	-	-	-	P	-	-	-
S-88-TT-26	01-17-88	Flagley Cty FL	1p	120	M	+	-	P	-	-	-
SWF-TT-8809-B KDL:894	01-17-88	Brevard Cty FL	-	137	F	+	+	P	+	+	+
S-88-TT-27	01-18-88	St. John's County FL	13	277	M	+	+	P	+	-	-
S-88-TT-28	01-18-88	Crescent Beach Fl	2	188	F	+	-	P	-	-	-
S-88-TT-29	01-18-88	Hammock FL	25+	260	M	-	-	P	+	-	-
S-88-TT-30	01-18-88	Ormond Beach Fl.	21	264	M	+	-	P	-	-	-
S-88-TT-31	01-18-88	Volusia Cty FL	8	242	F	+	-	P	+	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
S-88-TT-32	01-19-88	St. John's Cty FL	3	223	F	+	+	P	+	-	-
S-88-TT-33	01-19-88	St. Augustine Beach FL	1	163	M	+	+	P	+	-	-
S-88-TT-34	01-20-88	Atlantic Beach FL	6	235	M	+	+	P	+	-	-
S-88-TT-35	01-21-88	St. John's Cty FL	1p	129	M	-	-	P	-	-	-
S-88-TT-36	01-22-88	Ormond FL	3	231	F	+	-	P	-	-	-
S-88-TT-37	01-23-88	Flagler Cty FL	7	158	M	-	-	P	-	-	-
SWF-TT-8817-B KDL:1329	01-23-88	Brevard Cty FL	M	244	F	+	+	P	+	+	+
SWF-TT-8819-B KDL:1445	01-26-88	Brevard Cty FL	-	200	M	+	+	P	+	-	-
SWF-TT-8818-B KDL:1446	01-26-88	Sebastian Inlet FL	I	263	M	+	+	P	+	-	+
S-88-TT-38	01-27-88	Ormond FL	2	-	-	-	-	P	-	-	-
S-88-TT-39	01-28-88	Ormond FL	10	242	F	+	+	P	+	-	-
S-88-TT-40	01-29-88	St. John's Cty FL	-	110	F	-	-	P	-	-	-
S-88-TT-41	01-31-88	Ocean Beach FL	1	194	F	-	-	P	-	-	-
S-88-TT-43	02-01-88	Ormond FL	9	228	M	-	-	P	-	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
S-88-TT-44	02-01-88	Ormond FL	2	185	M	+	+	P	+	-	-
SWF-TT-8823-B KDL:1981	02-02-88	Brevard Cty FL	-	192	F	+	+	P	+	+	+
SWF-TT-8824-B KDL:1982	02-03-88	Indian River Cty FL	-	172	F	-	-	P	+	-	+
S-88-TT-46	02-06-88	Canaveral Nat'l Seashore FL	1[illegible]	241	F	-	-	P	-	-	-
S-88-TT-45	02-06-88	Volusia Cty FL	1[illegible]	233	M	-	-	P	-	-	-
S-88-TT-48	02-07-88	Volusia Cty FL	1[illegible]	140	F	+	+	P	+	-	-
S-88-TT-49	02-07-88	Volusia Cty FL	1	150	F	+	+	P	+	-	-
SWF-TT-8829-B KDL:2225	02-08-88	Brevard Cty FL	-	208	F	-	-	P	+	-	-
S-88-TT-50	02-09-88	Ormond FL	2	168	M	+	+	C	+	-	-
S-88-TT-51	02-10-88	St. John's Cty FL	-	166	M	+	+	P	+	-	-
S-88-TT-52	01-10-88	Flagler Cty FL	1	177	-	-	-	P	-	-	-
S-88-TT-53	02-10-88	St. John's Cty FL	1	184	F	+	+	P	-	-	-
S-88-TT-47	02-14-88	Ponce Inlet FL	-	194	F	-	-	P	-	-	-
S-88-TT-55	02-17-88	Canaveral Nat'l Seashore FL	7	227	F	+	+	C	+	-	-

Appendix 1 (cont'd.)

Dolphin ID	Stranding Date	Location	Age	Length (cm)	Sex	CHC	Heavy Metals	Necropsy	Histo-Pathol	Virol.	Micro-biol.
S-88-TT-56	02-18-88	Pineda Cswy FL	1[illegible]	240	F	+	+	P	-	-	-
S-88-TT-57	02-19-88	Canaveral Nat'l Seashore FL	7	205	M	+	+	C	+	-	-
S-88-TT-58	02-20-88	Mayport FL	22+	240	M	-	-	P	-	-	-
S-88-TT-59	02-20-88	Little Talbet Island FL	1	167	M	-	-	P	-	-	-
S-88-TT-60	02-25-88	Volusia Cty FL	I	135	F	-	-	P	-	-	-
S-88-TT-61	03-08-88	St. John's Cty FL	M	-	M	-	-	P	-	-	-
S-88-TT-62	03-08-88	Daytona Beach, Volusia Cty FL	-	185	M	-	-	P	-	-	-
S-88-TT-63	03-18-88	St. John's Cty FL	-	223	M	-	-	P	-	-	-
S-88-TT-64	03-29-88	Summer Haven, St. John's County FL	M	276	M	-	-	P	-	-	-
S-88-TT-65	04-07-88	Ponte Vedra, St. John's Cty FL	I	193	-	-	-	P	-	-	-
S-88-TT-66	04-09-88	Flagler Cty FL	-	100	-	-	-	P	-	-	-
S-88-TT-67	04-16-88	Flagler Cty FL	M	220	F	-	-	P	+	-	-
S-88-TT-68	04-21-88	St. John's Cty FL	M	-	F	-	-	P	-	-	-
S-88-TT-69	05-14-88	Florida	-	-	-	-	-	P	-	-	-

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May 19th, 1989

Mr. T.M. Foglietta
Chairman, Subcommittee on
Oversight and Investigations
Longworth House Office Building
Washington, DC

Dear Sir,

First, I want to let you know that I greatly appreciated the opportunity that you offered me to express my opinion before the Subcommittee on Oversight and Investigations about the 1987 dolphins strandings this last May 9.

In response to the question about the immediate actions that should be taken in regard with the strandings, I have the following suggestions: the severity of the event, the extent of the chrobic contamination of North Atlantic bottlenose dolphins by organochlorine compounds (OC) and particularly PCBs, the lack of data about other important key OC which usually accompany contamination by PCBs and which are more acutely toxic (dioxin, dibenzofurans) command a long-term monitoring program.

Before further defining such a program, here are some simple, relatively inexpensive actions: testing a dozen of menhaden fish from the North Atlantic for brevetoxin. These would include fish caught after the dolphins stranding and menhaden caught and frozen before the strandings occurred. If these easily available fish contain brevetoxin while dolphins strandings have stopped, this would strongly suggest that brevetoxin is normally present in menhaden and was not responsible for the strandings.

Since many years, carcasses of stranded marine mammals are stored in a walk-in freezer at the Boston Aquarium. Several times a year, necropsies of these animals are done and samples of major organs are kept frozen. I am not aware that these samples have been analyzed or that the results of these analyses have been released. Deep frozen organs are still suitable for analysis of OC and biotoxins. If brevetoxin was found, let say, in samples collected in 1985, its role in the 1987-1988 strandings would be weakened, at the very least.

The long term program should include: standard complete autopsy of each stranded carcass; systematic sampling of liver, kidney, blubber and fish contained in the stomach in order to be assayed for OC (including dioxin and dibenzofurans) and biotoxins; regular examination of captured animals for blood sampling in order a) to evaluate some essential immune functions (lymphocytes stimulation, phagocytosis) and b) to evaluate serum levels of keys OC compounds and biotoxins. Blubber biopsy of the captured animals should be taken to allow determination of these contaminants. Representative samples of fish from sensitive areas (near known offshore dumping sites) of the North Atlantic

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should be collected yearly to be analyzed for major OC. With these data, the effects of OC and biotoxins on the North Atlantic dolphins (and on the entire coastal ecosystem) could be determined correctly.

The second mandatory action is to determine the nature, origin, amount and location of sewage dumped offshore New Jersey in the recent months and, if possible, in the recent years. The nature of the containers, if there were any container at all, should be also known since some containers with highly toxic compounds could have ruptured many years after their dumping.

If such records do not exist, this is an im[portant lack in the offshore disposition of sewage and it is equivalent to accept the future occurrence of a similar event. It is also important to determine on which basis offshore dumping sites have been selected and if environmental impact studies have been undertaken before their selection. At the subcommittee hearing, the question whether dumping sites were used as food source for the dolphins or for their prey was not addressed. The composition of the "sludge" originating from incinerators and dumped offshore should be known.

Let me remind you that none of these data is present in Dr. Geraci's report. I do not understand the omission of all the data pertinent to dumping of industrial contaminants when a toxic substance was first suspected as cause of the strandings. Considering that lesions consistent with OC poisoning were found in most dolphins examined and that high levels of these compounds were found in all carcasses, I still do not see how contaminants have been ruled out as a cause of the strandings and how brevetoxin has been incriminated with such a confidence when the lesions that it causes, if it causes any lesions at all, are unknown and when this toxin has been found in 8/17 dolphins and 4 menhaden in amounts of which the significance is not clear.

Let me suppose for a moment that the 1987-1988 stranding was caused by a massive release of toxic man-made compounds from a dumping site in the North Atlantic in the summer 1987, what findings, exactly, would be expected? Would the 1987-1988 study have determined the nature of the compound? These questions should be addressed as well by a task force.

An evaluation of the impact of the strandings on the North Atlantic bottlenose dolphins population is necessary to have an idea of the number of these animals remaining in the North Atlantic. At the present time, I am not aware of any study that has been undertaken to assess if that population can eventually recover of these severe losses.

Finally, Mr. Evans stated that the report was submitted to the same review process as a scientific paper; this is not tenable. When a paper is sent to a scientific journal, the editor who is ideally objective and is not related to the researcher in any way first decides if the subject and the value of the paper are suitable for publication in his journal. After this first step, the reviewers whose expertise is pertinent to the subject are chosen by the editor in an objective way; Of course, the author of the paper does not know the reviewers. Also, when many investigators are contributors to the research, they sign the paper. Here, despite the large number of people involved in the study, one investigator signed. For instance, Dr. Baden, who did the brevetoxin analysis, did not not sign despite his major contribution.

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Even once a scientific paper has been published, its validity is only confirmed ultimately by the test of time. But before that, the reputation of the journal and, by corollary, of its reviewers are all factors suggesting that the content of the paper is correct. Here, many flaws are obvious in the process: the author of the paper is hired by the "editor" and a press conference (reviewed by whom?) is held before the written report is released (to whom?). At best, the report from Dr. Geraci is, as far as I am concerned, an interim, governmental, technical report.

The program or any other action should not involve a single individual, whatever his competence in marine mammals is. The problem considered here is a complex issue of of toxicopathology, of immunotoxicology, of toxicity of industrial and/or biological compounds, of oceanography and of marine biology and dolphins are a single component of the problem. Experts (immunologists, pathologists and toxicologists) in environmental toxicology (and I was going to add in political sciences and economy) should be and should have been consulted, and their report should be used and should have been used in any written or oral report since this problem is one of environmental toxicology. A report concerned with a problem of this magnitude should not have expressed the opinion of a single individual but rather should have reflected the consensus of an ad hoc task force.

Pharmaceutical companies take years, millions of dollars and hundreds of laboratory animals to determine the effects, toxic or therapeutic, of a new compound. Obviously, the problem that is dealt with here bears some similarities with the study of the effects of a (new?) toxic compound and consequently a definite answer to such a complex problem cannot be established without a long term work.

At least, the 1987-1988 strandings have revealed that very little is known about the location, nature, potential hazards and circulation of contaminants in north east American coastal waters. This is a potentially explosive situation. An opportunity is given to correct this.

For any additional information, please, do not hesitate to contact me.

Sincerely,

Daniel Martineau

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ADDENDUM

Potential members of a task force:

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cc P. Beland
B. Mckay

June 8, 1989

Mr T.M. Foglietta
Chairman,
Subcommittee on Oversights
and Investigations,
US House of Representatives
Committee on Merchant Marine and Fisheries,
Room 1334
Longworth House Office Building,
Washington
DC 20515 - 6230

UT NATIONAL
ÉCOTOXICOLOGIE
DU SAINT-LAURENT

ST. LAWRENCE
NATIONAL INSTITUTE
OF ECOTOXICOLOGY

Dear Mr Foglietta,

Thank you again for allowing me to express my views on a catastrophy that we would all wish never happened.

You will find herein the response to the question you asked me at the dolphin hearing on May 9. I apologize for taking so long, but I wanted to take time to send something constructive. I do hope that you will find this document helpful.

I would greatly appreciate bieng sent information regarding any new developments on this matter. Do not hesitate to contact me for any additional information that I may have.

Yours sincerely,

Pierre Béland
President

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THE 1987 DOLPHIN DIE-OFF :

WHAT COULD HAVE HAPPENED ?

HOW CAN WE FIND OUT ?

Response to a request by the Chairman,
U.S. House of Representatives Committee on
Merchant Marine and Fisheries

Hearing of May 9, 1989

INSTITUT NATIONAL
D'ECOTOXICOLOGIE
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Pierre BELAND, PhD
June 5, 1989

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INTRODUCTION

This document is a response to a request made at the May 9-10, 1989 hearings of the United States of America Congress Subcommittee on Merchant Marine and Fisheries. Part I is a speculative essay on possible scenarios that may have caused the dolphin die-off. It serves as a useful preamble to Part II, which responds to the Sub-committee Chairman's specific request for my views on what could be done to find out what happened to the East Coast dolphins in 1987.

The hearings did not modify my evaluation of the final report of the NOAA investigating team. Its chairman, Dr Geraci, did not provide new information. However, he indicated that his critics had not had access to the full set of data that were available to the team. This is certainly the case for me, inasmuch as I have not even seen any of the dolphins, their tissues nor the samples analyzed for contaminants, toxins or microorganisms. Nevertheless, a scientific report should stand on its own; alluding to additional but undisclosed data does not strengthen conclusions, nor does it clarify issues.

Basically, while the scenario retained by the investigating team appears unlikely, it was not sufficiently documented and demonstrated in the report. It is based on little hard evidence, and, as suggested by Dr Geraci's oral testimony - for example when he alluded to his knowing how a wild dolphin with a 'belly-ache' will behave - subjective thinking may have played a role in deriving conclusions. It is clear that the author of the report is unwilling to extrapolate from other mammals to dolphins when evaluating the effects of high levels of organochlorine contaminants. He does not however hesitate to extrapolate when evaluating the effects of brevetoxin. Yet, less can be found in the scientific literature about the effects of brevetoxin than about the effects of organochlorines.

It is true that the better known organochlorine contaminants, such as PCBs and DDT, have been present for at least 20 years in most marine mammals of the world. It is also true that marine mammalogists in the traditional sense have not paid much attention to the possible effects of high levels of these compounds. This stemmed partly from the lack of proper training, and partly from the difficulty of observing actual effects on ill-defined wild populations that normally ranged widely. Over the last 15-20 years, some evidence of deleterious effects of man-made pollutants has been produced from a number of populations of pinnipeds and one population of cetaceans. Recently, epidemiological studies on wild fish and birds, particularly in the Great Lakes basin, have shown that high levels of organochlorines have impacted wild animals and continue to do so.

It would not be wise to assume that the high levels of PCBs and DDT found in stranded East Coast bottlenose dolphins were not detrimental to their health. The NOAA report feels confident in assuming no effect when a few 'control' dolphins also showed high levels. It is important to remember that these so-called controls were also taken from the same environment. Captive animals that have been properly cared for (being fed clean food and given veterinarian treatment) since their capture some years past certainly do not constitute a convincing control group.

Finally, it would be wrong to assume that no toxic agents other than those listed in the report were involved. One must consider the possible role of many specific contaminants (including some that are more potent than PCBs and DDT), of natural or engineered viral and microbial agents, and of various toxic mixtures.

PART I

WHAT COULD HAVE HAPPENED TO THE DOLPHINS ?

1. What the report tells us :

Before finding out about what happened, Dr Geraci's findings can be used to orient the search.

It is apparent that the dolphins' immune system was down. This may have been a chronic condition resulting from their long-term exposure to immunotoxic compounds, as exemplified by their high PCB burdens. However, this response may also have been amplified by, and added to that resulting from acute poisoning by these same compounds. This would have occurred if the dolphins at that particular point in time had been reclaiming their fat reserves, thus exposing their vital organs to higher doses of organochlorine compounds.

Although the report alludes to this latter phenomenon as having occurred, it does not provide the data showing that the phenomenon did indeed occur (for example, by comparing blubber weight of stranded dolphins to that of control dolphins). Nevertheless, the evidence suggests that we look for some event that, either directly or through self-intoxication from blubber burden, would have triggered the demise of the dolphins. To a large extent, although not very strongly, the NOAA report asserts that brevetoxin exposure was such a trigger.

In the report, other pathological findings are also linked to the response one would expect from immuno-suppressed dolphins. Thus, bacteria and viruses identified in the tissues are opportunistic strains having taken advantage of weak or moribund animals. Overall then, the report emphasizes that the dying off was a multi-step and perhaps relativély slow process. This appears to be a reasonable assumption, as animals were beaching over a long period of time, sometimes with conditions that suggested various stages in the progression of a common health problem.

2. Where the report can be criticized :

In particular, my own testimony, augmented by those of Dr Martineau and other critics, pointed out that :

1) brevetoxin is but one possible, and perhaps even unlikely trigger;

2) in any case, if brevetoxin indeed was the trigger, PCBs and DDT already present in the dolphins tissues would then likely have been the bullet. The NOAA report should have at least emphasized this point, as the observed chronic liver lesions would have supported the hypothesis that organochlorine compounds had been active;

3. What the report does not tell us :

3) other potentially toxic compounds already present in the dolphins, but undocumented in the report, may have played a role similar to that of PCBs and DDT;

4) if point number 1 above is correct, then other possible triggers should have been investigated :

   a) it may turn out that some triggers would be potent enough to also act as a bullet either alone or in synergy with PCBs;

   b) some chemical triggers, when specifical- attacking tegument and mucosas (buccal, respiratory, anal), may have facilitated the work of bacterial and viral pathogens;

   c) some other biotoxin from an undocumented natural or man-made bacterial or viral strain may have been active.

4. What can be speculated :

As best as can be judged from the above, we are searching for a trigger and for a bullet leading to a somewhat prolonged process of disease and dying-off. We should therefore look for an event that occurred some time before the first deaths, say during the period January to June 1987. This event could have been a natural process (such as the brevetoxin hypothesis), or a man-made event such as the dumping of a chemical or biologically active substance at sea. Depending on where the dolphins were at the time the event affected them, this may have been a coastal or offshore occurrence.

PART II

HOW CAN WE FIND OUT ?

Where and when did it happen ?

1) Map the more likely route(s) of migration of the dolphins between January and June 1987; compile all reported observations of dolphins;

2) review all known coastal and offshore discharge and dumping events for that period;

3) look at NOAA satellite maps describing water currents and Gulf Stream eddies along the Florida to New Jersey coast;

4) review models predicting dispersion of various solutions away from dumping sites, in view of knowledge about solution characteristics (density, temperature, etc...) and oceanic site characteristics (stratification, presence of eddies, etc..);

5) interview crews of ships that were dumping offshore during the period January to June 1987 for observations of dolphins;

6) look at data from coastal monitoring programmes, such as the mussel watch programme, for unusual amounts and/or types of contaminants during that period;

What caused the die-off ?

7) re-examine the data gathered by the NOAA team. At this point in time, it is plausible to assume that the report did not include all analyses done, simply summarizing what were considered to be major and pertinent findings; Dr Geraci, in his testimony, alluded to the fact that critics, such as myself, had not had access to all available data;

   a) determine with figures the extent to which the dolphins were (or not) emaciated;

   b) examine lesions other than those summarized in the report;

   c) look at data on contaminants other than those tabled in the report.

8) reanalyze dolphin tissues for specific man-made contaminants and natural compounds. The search should be narrowed down on the basis of :

   a) toxic chemicals that are known to have been dumped during the target period ;

   b) particularly potent chemicals that are known to be present in this environment;

   c) clues gathered during a reexamination and reevaluation of dolphin tissues, lesions, bacteria and viruses;

   d) chemicals or toxins that are known to induce excessive bleeding (locally internally or systemic);

   e) chemicals that would insult skin and mucosas;

I apologize for the speculative and succinct nature of this text. It is intended to suggesting avenues for re-examining this unprecedented and worrisome event. If I can be allowed to make one additional recommendation, I would definitely advise that a team of experts be assembled to re-assess the NOAA report and to make recommendations regarding future action.

Pierre Béland
June 5, 1989

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Executive Director
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18 May 1989

Hon. Thomas M. Foglietta
Chairman, Subcommittee on Oversight and Investigation
U.S. House of Representatives
Committee on Merchant Marine and Fisheries
Room 1334, Longworth House Office Building
Washington, DC 20515-6230

Dear Congressman Foglietta

During the hearing on May 9 concerning the east coast bottlenose dolphin die-off, Congressman Pallone requested witnesses to ,rovide written suggestions for needed followup activity. Of pivotal importance at this moment is the security of analytical records and tissue samples, and steps should be taken to ensure that these sources of information are intact and are adequately protected.

The following additional comments are offered in response to Congressman Pallone's request.

First, it is striking that one of the most singular features of dolphins involved in initial strandings has been virtually ignored: the presence of extensive blisters and sloughing of the skin on animals that early reports characterized as appearing to have been "dipped in acid", and which Dr. Cassidy's report likens to chemical burns. These symptoms cannot be explained by the presence of PCBs in tissues, nor by ingestion of red tide algae. Further examination of possible causes of these burn-like lesions may provide an important component of the explanation for the entire event.

At this point we are data-rich and analysis-poor. From testimony offered on May 9, there seems to be agreement that the investigations conducted by Dr. Geraci and his colleagues have been performed in a competent fashion and the results are credible, though the interpretation and resultant conclusions of these results is questionable. These data can therefore be used to define specific questions that may require additional investigation to answer. But without the "focusing" that would be provided by a more complete evaluation of existing data, additional research may do little to provide an explanation for the event in question (though such research may well provide interesting information on broader questions related to status and trends within the marine environment).

To meet the need for more complete evaluation of existing data, it is suggested that existing data including results of individual chemical and pathological analyses be compiled, duplicated, and provided for review to individuals, agencies and institutions that will constitute the sort of diverse representation described in section 2(c) of H.R. 4189. It would also be highly desirable if the same information could be made generally available on a cost-of-publication basis. The emphasis should be upon identifying explanations that account for as much of the data as possible and that can be confirmed or disproven through reasonable tests. The initial emphasis should not be on additional research, because that will result in a long list of topics that individual scientists believe to be important, but that may not pertain directly to the problem at hand.

Proposed explanations might be evaluated through the following procedure:

a) solicit possible explanations for the die-off event that incorporate existing data and identify additional information or investigations needed to verify or disprove the hypotheses
b) subject proposed explanations to peer review with representation from both the academic and governmental sectors
c) convene a workshop of those who have examined existing data to evaluate possible causes of the event, and to define what additional information or action is needed to better resolve the question of cause

Implementing this approach should require no more than six months. At the end of that time, the Congress should be in a better position to determine the extent to which additional action is appropriate, and what that action should be. A number of agencies are capable of carrying out these procedures, but it is highly desirable that the exercise be open to a broad segment of the academic, commercial, and governmental communities and not be structured as the exclusive province of a few scientists and administrators.

I wish to congratulate the Subcommittee on its vigilence in monitoring investigations of the dolphin die-off, and will be pleased to offer any further assistance that may be useful in this process. With best wishes,

Sincerely

Melvin H. Goodwin

Melvin H. Goodwin, PhD
Coordinator,
Information and Extension Services

cc: F. Pallone

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